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TWENTIETH ANNUAL MEETING

OF THE

AMERICAN GASTRO-ENTEROLOGICAL ASSOCIATION

HELD AT THE

HOTEL TRAYMORE, ATLANTIC CITY, N. J.

April 30, and May 1, 1917

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Walter J. Dodd	374 Marlborough St., Boston, Mass.

CONSTITUTION AND BY-LAWS
OF THE
AMERICAN GASTRO-ENTEROLOGICAL ASSOCIATION.

CONSTITUTION.

Article 1. This Association shall be known as the American Gastro-Enterological Association.

Article 2. The object of this Association shall be the promotion of investigation of the normal and pathological conditions of the digestive organs.

Article 3. Membership in this Association shall be of three kinds: (*a*) Honorary, (*b*) Corresponding, (*c*) Active

(*a*) Honorary membership shall be restricted to such as have attained an international reputation by their published works and have scientifically furthered the subject of gastro-enterology. Their nomination shall take place at the annual meeting, upon the written suggestion of at least six active members and on the recommendation of the Council of the Association at the next annual meeting. Their election shall be by unanimous vote of the members present.

(*b*) Corresponding membership shall be restricted to foreign scientists in recognition of meritorious work within the scope of the Association. Candidates for such membership shall be proposed by at least six active members and receive the recommendation of the Council at the next annual meeting. Their election shall be by unanimous vote of the members present.

(*c*) Active membership shall be restricted to American and Canadian investigators and practitioners who have published meritorious work in normal or pathological anatomy or physiology, in medicine or surgery of the digestive canal and its secretory appendages, and who enjoy an unimpeachable moral standing in the medical profession.

Names of candidates for active membership must be: (1) Proposed by two active members who are not members of the Council, such proposal to be submitted to the Committee on Admissions and Ethics, which should report to the Council as soon as possible; (2) Must be recommended by the Council and (3) must receive a majority of the votes of active members present at the annual meeting of the Association.

The election of active members shall take place at the annual meeting of the Association and shall be by ballot.

It shall be the duty of the Secretary to notify active members of all nominations that are to be submitted to the Association at the annual meeting.

ARTICLE 4. The officers of the Association shall be a President, two Vice-Presidents, a Secretary and Treasurer, the latter two offices being vested in one; also three specially chosen active members to be known as Councilors, who, together with the officers, shall constitute the Council of the Association. The said Councilors shall be elected as follows: one for one year, one for two years, and one for three years.

ARTICLE 5. There shall be a Committee on Admissions and Ethics. It shall consist of the President and the Secretary and of three other members, not members of the Council. These members shall be elected at the annual meeting at which this Amendment shall be adopted for the following terms: one for three years, one for two years and one for one year. At each subsequent annual meeting one member shall be elected for the term of three years to fill the annual vacancy. No member of the Committee shall be eligible for re-election until after one year shall have elapsed from the end of his term of service. It shall be the duties of the Committee on Admissions and Ethics to examine the merits of a candidate, to investigate any charges made against a member, and to report the results of the examinations and investigations to the Council in writing in the shortest possible time. It shall also be the duty of this committee to study the active work in science and practice of gastro-enterology accomplished in this country and Canada and to invite the meritorious workers to join this Association.

ARTICLE 6. The election of officers, Councilors and of the members of the Committee on Admissions and Ethics shall take place at the annual meeting and shall be by ballot.

ARTICLE 7. Vacancies in the offices occurring in the interval between the annual sessions shall be filled temporarily by the Council.

ARTICLE 8. The annual meeting of the Association shall be held at a time and place to be decided by the Council.

ARTICLE 9. The President shall be the chairman of the Council.

ARTICLE 10. There shall be annually two meetings of the Council; one shall take place at the call of the President, shortly after the annual meeting of the Association; the other shall take place at least four weeks previous to the annual meeting. It shall be within the discretion of the President to conduct this latter meeting by correspondence.

ARTICLE 11. The Council shall manage the affairs of the Association in accordance with the Constitution and By-Laws, and its minutes shall be reported to the Association at the annual meeting.

ARTICLE 12. This Constitution may be amended by a two-thirds vote of all the active members at any annual meeting, provided that notice of proposed amendments has been given in writing to all the active members of the Association by the Secretary, and provided further that such proposed amendments shall have been submitted to the Council and held over for one year.

Article 13. Any member failing to attend three consecutive annual meetings without an excuse acceptable to the Council shall be dropped from the roll. The Secretary, however, shall be required to call the attention of a delinquent member to the facts in his case previous to the third meeting.

Article 14. This Association adopts the Code of Ethics of the American Medical Association. Charges of advertising, of publishing knowingly false scientific statements, of actions unbecoming a gentleman and a high standing physician, etc., shall be investigated by the Committee on Admissions and Ethics. The accused shall have liberal opportunities to clear himself of the accusation. The Committee shall report its findings to the Council, and on the recommendation of this body an offending member may be expelled by a three-fourths vote of those present at the annual meeting.

BY-LAWS.

Article 1. The President and Vice-President shall discharge such duties as are implied by their respective offices. The President shall preside at all sessions of both Council and Association.

Article 2. The Secretary shall attend to the usual clerical duties of the Council and Association. As Treasurer, he shall keep a record of the payments and arrearages of dues, and report on both at the annual meeting.

Article 3. The Council shall report its transactions to the annual meeting of the Association. It shall superintend the publication of scientific papers, but shall not appropriate for such printing an amount exceeding the sum of $100, without the vote of the Association. The order of business shall be arranged by the Council before the annual meeting. It shall pass upon the eligibility of candidates for membership.

Article 4. The yearly dues of active members shall be five dollars, payable in advance. Any member whose subscription shall be in arrears more than two years shall be reminded of the fact by the Treasurer, in writing; in event that payment be not then made he may, on vote of the Council, be dropped from the roll of the Association. It shall be the duty of the Treasurer to report all members who are in arrears to the Council.

Article 5. When sufficient money is in the treasury the membership dues may be omitted when decided by the Council.

ARTICLE 6. These By-Laws may be amended, repealed or suspended by a two-thirds vote of the members present at any meeting of the Association.

ARTICLE 7. The quorum of the Association for the transaction of business, but not for the reading of papers, shall consist of seven active members.

ARTICLE 8. All titles of papers submitted for reading at the annual meeting shall be sent to the Secretary not later than four weeks before the date of the meeting.

ARTICLE 9. A typewritten abstract of not over three hundred words, of every paper to be read, must be sent to the Secretary at least four weeks before the annual meeting. It shall be the duty of the Secretary to send to each member of the Association a complete list of the papers to be read at the annual meeting, with their full titles, at least two weeks before the annual meeting.

ARTICLE 10. Papers submitted to be read before the Association shall not have been previously published or read elsewhere. Papers shall not exceed the limit of fifteen minutes without the consent of the majority of members attending.

PRESIDENT'S ADDRESS.

A RESUME OF THE PROGRESS IN GASTRO-ENTEROLOGY DURING 1916.

In the busy strenuous lives the present generation are living, when every hour of each day is filled to the overflowing with exacting duties which brook no delay, little time is given to us for noting in detail the progress which is rife everywhere around us. Is it not wise, for that reason, to pause once a year to take account of stock, as it were, to make that which is good our own, and to cast aside into oblivion that which seems unworthy either from a scientific aspect or for practical purposes? Therefore I will ask you to turn aside for a moment from the consideration of the good things in anticipation, to survey with me the field of progress for the last year so that we may decide upon those things which hold some promise for the future.

My predecessor in this chair[1] in his address called attention to two conditions which had not been presented to us in that way before: the first was the consideration of acute gastrectasis, not from the standpoint of the text book type but rather the atypical cases following, usually, abdominal operations.

The second condition which he described was an unusual type of acid regurgitation in which there was relaxation of the cardia as well as of the pylorus so that whenever peristalsis occurred a small portion of the meal was forced back into the gullet while the major part was passed down into the pyloric pouch.

Einhorn[2] in his paper entitled, "The Duodenal Tube as a Factor in the Diagnosis and Treatment of Gall-Bladder Disease," takes us a step farther in the development of the use of the duodenal tube when he points out his treatment of diseases of the gall-bladder by introducing through it solutions of argyrol and ichthyol, thus applying the medicament to the duodenum just above Vater's ampulla.

Cheney,[3] acting upon the suggestion of Einhorn, uses the duodenal tube to introduce a twenty-five per cent. solution of sulphate of magnesia into the duodenum and assures us of the remarkably beneficial results from its use. This use of sulphate of magnesia was first suggested by Meltzer.[4] Cheny believes it has the effect of relaxing the sphincter of the common duct permitting the ejection of bile and even of allowing a calculus of moderate size to pass out. This would appear rational enough to merit a practical test of its usefulness.

Garnette,[5] in a recent paper, describes what appears to be an entirely new treatment for pernicious vomiting of pregnancy and reports two cases successfully treated. The theory of his treatment is based upon the belief that pregnant women develop an antigen which protects them from the ravages of the toxins developed during the course of pregnancy. He believes that those patients who are attacked with pernicious vomiting have failed to develop the specific antigen. In the first case all of the known measures were tried without effect and he finally conceived the idea of transfusing the patient with the blood of a normal post-partum woman. This patient was relieved of the exhausting vomiting and went on to full term, being delivered of a healthy child. A second case some months later was treated in the same manner with a like favorable termination. If further experience with this method established its validity Dr. Garnette has given to the medical profession a most valuable and at times life-saving method of treatment. This would naturally lead us to wonder whether a similar procedure might not prove equally efficacious in nephritis of pregnancy and of scarlet fever.

Stengel and Hopkins[6] describe a new method for determining the intra-gastric temperature in man, with some observations on its variation after the ingestion of hot and cold liquids, and during digestion. They describe their apparatus which consists of a thermocouple of copper and constantin wires, one pair of ends being placed in a thermos bottle and the other pair being soldered together and run through an Einhorn duodenal tube at the end of which they are made secure by solder within the small perforated metal bulb. The thermocouple is then joined to a mirror galvanometer and a potentiometer, the reading being taken in microvolts. Full meals were taken alongside of the tube and the fluoroscope used to determine the location of the bulb, thus enabling them to observe variations in temperature between the pyloric and the fundic areas.

The great difference in temperature between the fundus and the pylorus after taking cold drinks or foods and the length of time required to resume the normal temperature are interesting as confirming the recent researches of Grutzner.

Rosenow[7] has presented further studies on the causation of peptic ulcer in a paper entitled "Causation of Gastric and Duodenal Ulcer by Streptococci."

He goes on to say that streptococci have a characteristic affinity for the stomach and duodenum, have been repeatedly isolated from the various focci of infection in patients with ulcer, and from the ulcer itself. They tend to disappear from the circulation and do not commonly produce marked lesions elsewhere. They have been isolated from ulcers in animals, and ulcer has been produced by reinfecting animals with them. Filtrates of these cultures show no tendency to produce ulcer. He concludes that the necessary requirements have been fulfilled to warrant the assumption that the usual ulcer of the stomach and of the duodenum in man is primarily due to a localized hematogenous infection of the mucous membrane by streptococci.

Cellar and Thalheimer[8] in an article entitled "Bacteriological and Experimental Studies on Gastric Ulcer" take issue with Rosenow.

These authors cite Rosenow's work on the production of acute and chronic ulcers in cats and dogs, and give his conclusions. Their experiments were made with a view of testing the truth of Rosenow's deductions. They used the same technique which he used.

As a result of their studies they say that it is impossible to decide definitely whether or not the gastric lesion produced by the injection of streptococci are to be considered ulcers. The superficiality of the rabbit lesion following the injection of streptococci by the intravenous route, as well as the entire absence of inflammatory reaction in the deeper gastric tissues leads them to believe that these defects are not analogous to the chronic gastric ulcers seen in the human stomach. The promptness of the healing of the lesions in cats tends to strengthen this conclusion.

Wilemsky and Geist[9] in a paper entitled "Experimental Studies in the Production of Chronic Gastric Ulcers," agree with Rozenow in the conclusion that streptococci may be a factor in the production of these ulcers.

Spencer, Meyer, Rehfuss and Hawk[10] present a paper entitled "Direct Evidence of Duodenal Regurgitation and Its Influence Upon the Chemistry and Function of the Normal Human Stomach," in which they discuss Boldyreff's theory, "The Self-Regulation of the Acidity of the Stomach Contents."

Their conclusions are summed up in twelve distinct observations of which the final one is that the regurgitation of duodenal contents into the stomach is but another of the protective functions of which the body furnishes so many examples and has for its purpose the defense of the small intestines from irritants.

Mann[11] has pointed out that animals dying after the removal of both adrenals show acute ulceration of the gastric mucosa in the majority of cases, and suggests the possibility of the influence of the adrenals on the production of peptic ulcer.

Cook, Rodenbaugh and Whipple[12] give the results of their investigations on intestinal obstruction;. a study of non-coagulable nitrogen in the blood. This paper deals with the analyses of the blood in intestinal obstruction and other acute intoxications. The work of these authors seems to establish the fact that intestinal obstruction is associated with an increasing amount of non-coagulable nitrogen in the blood and that the more acute the intoxication the more rapid and the higher the rise in the blood, of non-coagulable nitrogen. They point out that the transfusion of dextrose solution often benefits intestinal obstruction and depresses the level of non-coagulable nitrogen in the blood, and where this does not occur a fatal termination may be expected.

Crile[13] in his paper entitled "Methods and Results in Surgery of the Stomach and Intestines," shows that the principle cause for the high mortality in resection of the stomach and intestines was acidosis, the reserve alkalinity of the body having been lessened to the minimum.

Hull[14] published a paper entitled "The Influence of Milk and Carbohydrate Feeding on the Character of the Intestinal Flora" in which, after discussing the results of many experiments, he has drawn the conclusion that the mere feeding of bacillus Bulgarius does not lead to the acclimatization of the organism in the intestines even for a short time.

Wall[15] has devised a new operation for gastropexy and cystopexy which consists in passing three or four sutures from deep in the anterior wall of the stomach up and fixing them to the peritoneum and fascia in the upper part of the median incision. It is similar to the method of Duret and Rovesing except that it has the advantage of allowing folding in two directions, longitudinally and transversely.

Dunn[16] points out that it is comparatively easy for the surgeon to find the hidden stomach ulcer by using his new route of approach ('inter-colo-epiploic route') which is especially useful when inflammatory adhesions must be separated.

Quain[17] describes a new instrument for the application of the sewing machine stitch in gastro-intestinal surgery. He goes on to state that absolute hemostasis is necessary for safe and successful gastro-intestinal surgery. Attention is called to the double interlocking through and through running suture made by the ordinary sewing machine. Whenever this type of suture can be applied to living tissues bleeding is practically impossible. Quain's instrument renders this procedure possible and easy. A detailed description of the instrument is given in his published article. He has used this method in seventeen gastroenterostomies as well as in several bowel resections, and only an occasional clot of blood was found in the stomach. He claims that by this method he has been able to save nearly half the time previously employed in gastroenterostomy.

Harter[18] describes a most remarkable method for securing complete asepsis in gastro-intestinal operations, which consists of passing a jet of steam through the bowel for from fifteen to thirty minutes and states that by this means all of the bacteria are killed and that no change was noted in the bowel wall. One would feel that such a procedure should be well worked out upon subjects already dead before giving it a practical application upon the living.

Alvarez[19] in his paper entitled "Differences in the Irritability and Latent Period of Different Parts of the Wall of the Stomach," describes the results of experimental work on rabbits, cats, and dogs, and a few experiments upon the human stomach. He found the lesser curvature and that part near the cardia more irritable than the fundus and body of the stomach.

The tendency of the antrum to respond as a whole when stimulated at any one spot may have some clinical interest. As the pylorus is more irritable than the rest of the antrum, it is not surprising that it should often be closed so tightly when this part of the stomach is irritated.

The author agrees entirely with Meltzer that it is quite hopeless to attempt to influence the stomach therapeutically by intragastric of abdominal electricity.

Ginsburg, Tumpowsky, and Hamburger (20) have published an excellent paper entitled "The Newer Interpretation of the Gastric Pain in Chronic Ulcer." They discuss the latest theory, suggested by Hertz, that the cause of the pain is in the sudden increase in intragastric tension probably due to exaggerated peristalsis in a hypertonic organ. Hertz believes that excess acid passing into the duodenum prevents relaxation of the pylorus or that the ulcer exposes more nerve endings in the stomach walls, the irritation of which by the acids causes pyloric spasm and intensified gastric tonus. The authors are inclined to accept this view and draw the following conclusions: "First, the finding of strong contraction of the stomach accompanying the pain of gastric ulcer seems to confirm the idea that the pain is due to tension. Second, the marked hunger contractions cause pain in a hyper-irritable condition of the stomach by increasing intragastric pressure. Third, the conception that gastric pain is due to tension will explain many obscure conditions simulating gastric ulcer, viz., achylia gastrica, chronic appendicitis and gall-bladder disease. Fourth, hyperacidity alone may be a factor by reflexly causing hypertonus, hyperperistalsis and pylorospasm allowing greater tension to be performed. Fifth, the subjective relief of pain by alkalies does not necessarily prove that acid is the cause of pain, but may be interpreted on the basis that alkalies prevent the development of pain producing hypertonus by neutralizing the causative of such hypertonus, *i. e.*, pain. Sixth, hydrochloric acid in the strength that it may occur in the stomach, causes no appreciable effect.

Friedenwald and Kieffer[21] discuss the diagnosis of cancer of the stomach in a paper entitled "The Value of the Quantitative Elimination of Dissolved Albumen in the Gastric Contents in the Diagnosis of Cancer of the Stomach."

They refer to the work of Wolff and Jughans in connection with the estimation of the soluble albumen in the gastric extract which they claim is of great value in the diagnosis of gastric cancer. Smithies confirms this view. They point out that this test is only useful in the diagnosis of the disease when there is an absence of free hydrochloric acid in the gastric contents, and then only when the question of even traces of blood can be eliminated, and in the absence of all retained food residue or of swallowed saliva or sputum. They claim their test has its greatest significance in the diagnosis between simple and malignant achylias. A positive reaction rarely occurs in malignant growths in the abdomen not involving the stomach.

Lerche,[22] following the thought expressed by Mayo some time previously, expresses the belief that chronic irritation from the ingestion of hot fluids is an important predisposing cause of cancer of the esophagus and stomach, and supports this view in his paper entitled "A Contribution to the Etiology of Cancer of the Stomach and Esophagus." His conclusions, which are somewhat lengthy, are well drawn and have the support of logical reason.

Elliott and Henry[23] have presented some observations upon surgical work with the army in France with special reference to the stomach in a paper entitled "Traumatic Ulcers." In this paper they point out a fact which has been generally recognized for a long time—that any wound of the intestinal tract is less likely to prove fatal if not repaired at once.

They point out that while the chief danger in intestinal wounds is from peritonitis, in stomach wounds it is less to be dreaded, as frequently adhesions seal the aperture and the gastric contents that do leak out are not especially sceptic. However, the real danger in stomach wounds is from secondary hemorrhage due to erosion of the injured tissue by the gastric juice. Immediate hematemesis and melaena are rare, as the bleeding is usually intra- or retro-peritoneal. But these symptoms usually appear during the second week, and are then due to an extending ulcer of the stomach or duodenum. These ulcers are the exact counterpart of the non-traumatic ones.

Swete, Peet and Hendricks[24] published a very excellent paper entitled "High Intestinal Stasis," in which these authors describe a series of experiments to determine the cause of death in high intestinal obstruction, the clinical picture of which suggests a grave constitutional disturbance of a toxic nature. Their results support Draper's idea that the toxin is a normal product of the duodenum which under normal conditions is neutralized by the jejunum. It is believed that high intestinal obstruction is due to the profoundly toxic properties that have been formed in the proteose stage of protein digestion.

The two prominent features of these authors' experimental work are: 1st, the added demonstration of the fact that a gastro-enterostomy opening does not function in the presence of a normal pylorus. 2nd, the explanation of the similarity between acute pancreatitis and obstruction; they are alike because they are both essentially the same thing, an intoxication with the toxic products are protein cleavage, which in pancreatitis is. certainly due to proteolytic ferment of the pancreas, and in high obstruction it is probably the same toxin produced by the same ferment.

Case[25] discusses the Roentgen ray findings in diabetes and points out that there is a striking relation between the severity of the disease and the degree of ilial stasis. Incompetency of the ileo-cecal valve was a common finding. Adhesions of the terminal ilium, stasis in the cecum and evidence of disease of the appendix were somewhat more frequent in the severe cases than in the mild.

The paper is intended as a contribution to the study of intestinal toxemia as one of the probable causative factors of diabetes.

Carman[26] in a paper entitled "The Roentgenologic Diagnosis of Duodenal Ulcer," gives his experience for the past year in this field. He points out that deformity of the duodenal shadow is a valuable sign in the diagnosis of duodenal ulcer. He used serial radiography during this period with satisfactory results and with fewer incorrect diagnosis.

In addition to duodenal deformity the author considers gastric hypertonus, hyperperistalsis, hypermotility with a six-hour residue valuable diagnostic signs of duodenal ulcer.

Saralegui[27] discusses the subject of the diagnosis of intestinal obstruction by means of radiography. He comes to the conclusion that when the pylorus is permeable and there is no other lesion which detains the ingesta in the stomach he can make a diagnosis of obstruction in the small gut if nine hours after a meal remnants of bismuth are still in the stomach.

Brown,[28] in a most interesting and convincing paper discusses the subject of intestinal intoxication, and gives the results of his observation upon three cases of chronic eczema and urticaria, one of pruritus and two of bilious headache and one of choroiditis.

He concludes his paper by saying that in the study of these cases it is hard to escape the conclusion that in the intestines certain substances are produced which are possessed of specific action associated with a hypersensitiveness to certain proteins, and that therapeutically a marked improvement and even complete cure may be brought about by the elimination of these substances from the dietary.

Dragstedt,[29] in considering the physiology of the stomach discusses the condition of the gastric juice in gastric and duodenal ulcers. He begins his paper by saying, "The present work was undertaken to learn the effect of gastric juice upon the rate of healing of acute gastric and duodenal ulcers. It is well known that the lesions of the gastric and duodenal mucosa heal readily in the presence of active gastric juice. Small abrasions of the stomach mucosa, such as those following at times the administration of a stomach tube, are common, occasion no discomfort to the individual, and in a majority of cases heal without further sequela. Surgical operations in which there is a maximum insult to the mucosa cells are not commonly followed by untoward effects to the digestive activity of the gastric juice."

After discussing at some length the results of his experiments he draws the following conclusions: "1, The digestive activity of gastric juice is not the important factor in the delayed healing of acute ulcers of the stomach and duodenum and the consequent formation of chronic ulcers.

2. Ulcers produced by local injection of solutions of silver nitrate become subsequently infected with organisms probably from the alimentary tract."

Garbat[30] gives a very able discussion of the convalescence of typhoid in his paper entitled "Duodenal Cultures in Typhoid Fever as a Means of Determining Complete Convalescence."

He goes on to say: "Some statistics regarding typhoid fever will show the object of this work." 1. Of every ten thousand apparently perfectly healthy residents; 2. Three are typhoid carriers.

Fifty-five per cent. of all typhoid cases are due to carriers, either directly or indirectly * * * * *

3. Park has estimated that one in every five hundred adults who never knowingly had typhoid, is a carrier.

4. Of every hundred typhoid cases, from three to six become "carriers."

The author goes on to describe the method of collecting the duodenal contents through the Einhorn duodenal tube and described the manner in which the examinations are to be conducted. He closes his paper with the following conclusions: "Cultures from duodenal contents (bile) removed by means of the duodenal tube seem to be a more reliable and simpler method for the detection of typhoid bacilli than the stool examinations."

Meltzer[31] read before this society last year a most interesting and instructive paper, which we all remember, entitled "The Disturbance of the Law of Contrary Inervation as a Pathogenetic Factor in Diseases of the Bile Ducts and the Gall Bladder."

In this paper the author discusses at some length the physiological activity of the gall bladder and bile ducts, and compares it to the urinary bladder. He points out the differences in the functioning of the two. After discussing the more commonly recognized physiological activities of the gall bladder he draws attention to some facts which have hitherto escaped the attention of the general practitioner. He shows that bile salts, as well as pigment, are reabsorbed from the intestines to be stored up in the gall bladder and shows the advantages of such a procedure by telling us that the presence of bile in the intestines at such times as it is not needed for digestive processes may exert a deleterious influence upon the gut. From this he draws the conclusion that in this way the gall bladder exerts a protective action. He also calls attention to the fact that the bile in the gall bladder is much more concentrated than that which is present in the bile ducts.

He shows that this process of storing up in the gall bladder, while a necessary physiological process, yet may become, and often does so, a source of danger to the economy.

The author discusses the infection of the bile by pathogenic micro organisms and asks the question, what prevents their more frequent occurrence. He says there are two elements in this protective influence. One is keeping the mucus membrane of the gall bladder and bile ducts in a healthy normal condition. The other factor is in preventing prolonged stasis of bile in the gall bladder and biliary passages, but lays especial stress upon the latter.

In conclusion the author suggests the advisability of eating frequent small meals which are composed of foods which readily furnish peptones and albumosis to the duodenum. He suggests that this should be especially observed in those diseases in which the bile is apt to contain positive organisms, such as typhoid and the like, since in this way the bacteria-laden bile may be discharged at frequent intervals, thus preventing the development of diseases of the biliary passages.

White's[32] paper entitled, "Some Roentgen Ray Evidence in Gastro Intestinal Lesions," was timely, since there is a tendency among general practitioners especially, to permit the radiographer to make, unquestioned, their diagnosis for them.

The author has considered the fallacies encountered in radiographic studies upon the various parts of the gastro-intestinal tract, and concludes his paper as follows: "The new facts given us by this method are abundant and bewildering at first, and bring a few new sources of error and call for new judgments. An esophageal pouch, an old cancer of the stomach, a calcified gallstone are in a very different class from early cancer, cholesterin stones and small intestinal adhesions. In addition there is at times a difficulty in distinguishing mere personal peculiarities from actual signs of disease. But finally, in spite of these difficulties and limitations the method has immense value in diagnosis, both present and future."

REFERENCES.

[1] Transactions of American Gastro-Enterological Association, 1916.

[2] Transactions of the American Gastro-Enterological Association, 1916.

[3] *American Journal of Medical Sciences*, April, 1917.

[4] *American Journal of Medical Sciences*, April, 1917.

[5] *New York Journal of Obstetrics*, to be published at an early date.

[6] *American Journal of Medical Sciences*, January, 1917.

[7] *Journal of Infectious Diseases*, 1916, XIX, 333.

[8] *Journal of Experimental Medicine*, 1916, XXIII, 791.

[9] *Journal of American Medical Association*, 1916, LXVI, 1382.

[10] *American Journal of Physiology*, 1916, XXXIX, 459.

[11] *Journal of Experimental Medicine*, 1916, XXIII, 203.

[12] *Journal of Experimental Medicine*, 1916, XXIII, 717.

[13] *Buffalo Medical Journal*, 1916, LXXII, 55.

[14] *Journal of Bacteriology*, 1917, II, 47.

[15] *Surgical Gynecology and Obstetrics*, 1916, XXII, 485.

[16] *American Journal of Surgery*, October, 1916.

[17] Transactions of the Western Surgical Association, St. Paul, December, 1916.

[18] *Beitr. Z. Klin. Chir.*, 1916, XXLX, 475.

[19] *American Journal of Physiology*, 1916, XLI, 321.

[20] *Journal of American Medical Association*, 1916, LXVII, 990.

[21] *American Journal of Medical Sciences*, 1916, CLII, 321.

[22] *Surgery, Gynecology and Obstetrics*, 1916, XXIII, 42.
[23] *British Medical Journal*, 1916, I, 532.
[24] *Annals of Surgery*, 1916, LXII, 720.
[25] *Journal of the American Medical Association*, 1916, LXVII, 858.
[26] *American Journal of Roentgenology*, 1916, III, 252.
[27] Rev. Asoc. Med. Argent., 1916, III, 86.
[28] *American Journal of Medical Sciences*, 1916, CLII, 845.
[29] *Journal American Medical Association*, February 3, 1917.
[30] *Journal American Medical Association*, November 18, 1916.
[31] *American Journal of Medical Sciences*, April, 1917.
[32] Transactions American Gastro-Enterological Association, 1916.

A BARIUMIZED TEST MEAL WITH COMBINED FLUOROSCOPIC AND CHEMICAL STUDY AS A MEANS OF RAPID STOMACH DIAGNOSIS.*

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The chemical processes of gastric digestion have been studied very minutely and extensively for a number of years, these studies being largely limited to the phenomena associated with the so-called Ewald breakfast. This test meal, it is fully recognized, does not call out and therefore does not measure the reserve powers of gastric digestion and is consequently of a somewhat limited value in the general field of gastric diagnosis. Such an enormous literature has, however, been built upon its widespread clinical use that it must be regarded as of decided value for comparative purposes, while fully realizing its limitations. It is undoubtedly true that an average normal stomach will usually behave in a certain manner under the stimulus of the Ewald test breakfast and important information concerning secretory and even motor conditions may be obtained.

It occurred to me that if a rapid fluoroscopic method could be combined with this test meal that much preliminary information could be quickly obtained. Such an examination made under favorable conditions with an empty stomach would in many cases furnish all the X-ray data necessary for diagnosis. This could be done in about an hour's time when a quick survey of the case was desirable, in conjunction with and during the progress of other preliminary investigations, such as taking the anamnesis, physical, blood and urine examinations, etc., and would rapidly place the clinician in possession of what might be called a skeletal outline of the most important motor secretory and topographical facts. It is, of course, taken for granted that further fluoroscopic or even radiographic methods will be used when required for the purposes of a satisfactory working diagnosis.

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*Read before the American Gastro-Enterological Association at Atlantic City, N. J., April 30th, 1917.

It is well known that the most divergent opinions prevail in European and some American clinics in regard to the relative value of X-ray pictures and fluoroscopic examinations. The controversy applies mainly, if not entirely, to certain aspects of diagnosis in the field of internal medicine. In regard to surgical lesions or injuries, for example, of the bones, the X-ray plates are absolutely essential for clearness of detail and permanence of record which may often be important from a medico-legal viewpoint. In the early stages of pulmonary tuberculosis it is true that the photographic plates will show details which the fluoroscopic screen cannot reveal, although slight opacities shown on the screen will often quickly guide the clinician to quite early lesions. In regard to the heart, the screen will generally give all the information needed. In the stomach, and in fact in the entire alimentary tract, very competent observers believe this also to be the fact. For instance, Dr. J. T. Case,* than whom few are better qualified to judge, says: "In the study of the alimentary tract, however, fluoroscopy is the method of choice or, rather, we may say, the method which gives us by far the greater yield of valuable information concerning the case. The roentgenogram furnishes a representation of static conditions, the shape (at the moment) and the position; but, of far greater importance in the majority of cases, roentgenoscopy furnishes data concerning mobility, motility, coincidence of pressure pain points with filling defects, spastic manifestations in the stomach and other questions relating to function, which, by roentgenograms alone, either cannot be shown at all, or only with difficulty and at considerable expense. After a large experience in roentgenographic and roentgenoscopic examinations of the alimentary tract, I can truly say that with the exception of gall-stone and duodenal ulcer cases, in comparatively few instances have the roentgenograms added any material fact of importance to the fluoroscopic findings. In the average patient fluoroscopy of the stomach is quite satisfactory, no plates being needed except for the recognition of possible gall-stones."

As representing at least one aspect British medical opinion, Barclay says**: "We have the choice of radiography and radioscopy as our method of examination, and in my opinion there is

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*Medical Clinics of Chicago, July, 1916, p. 205.
**The Alimentary Tract, 1915, p. 6.

no comparison. Radioscopy (fluoroscopy) is the method of necessity. The examination should be undertaken with the patient standing with his abdomen against the fluorescent screen, and the passage of the first mouthfuls should always be carefully observed from the lower third of the oesophagus till the lower part of the stomach is outlined. * * * Radiographs of the stomach are of comparatively little use except for demonstration purposes; they represent the picture at one particular moment only, and give little indication as to how the stomach received the food, etc. Radiographs are, therefore, expensive, and in many cases unnecessary luxuries except for demonstration purposes, but a good radiogram of the pars pylorica will often reveal more detail than can be made out on the screen."

Certain details, as for instance, in suspected pyloric diseases, filling and budding defects, etc., can perhaps be better studied by radiographs than by fluoroscopic examination, but the fact seems to be that all the help needed in the majority of cases for diagnostic purposes can be obtained by the screen and the filling of the stomach, which is of the utmost importance, together with the peristaltic phenomena can only be satisfactorily studied in this way.

In attempting to combine fluoroscopic study which would demonstrate topography and motility with the chemical study of gastric secretion, certain questions presented themselves for solution. The most important of these related to the influence of the barium or other opaque metallic salt upon the determination of the acid content by the usual methods of titration. These questions we attempted to solve in my clinical laboratory. It was demonstrated in the first place that barium did not by any process of chemical reaction modify the quantity of free HCl. If, for example, a solution was shown to contain 60° of free HCl and sufficient barium added to make it very turbid, the titration would give exactly the same result as before the barium was added. The barium it was found could not be removed by ordinary filtration so as to give a clear solution, but the color reaction was sharply defined and the presence of the barium turbidity rather to my surprise offered no obstacle of practical moment to correct titration. This was found to be true whether proteids were present or not. At this point the way seemed clear to combine these two methods of clinical study. I soon found, however, that when considerable quantities of barium up to one or two ounces were added to a test meal that the secretion of the oxyntic glands, as compared with that which occurred with barium free test meals was partially and sometimes almost completely inhibited. I had satisfied myself by experiments in vitro that this lowered acidity was not due to any chemical effect of the barium of the gastric contents. The only conclusion left was that in some way the barium depressed or inhibited the secretory functions of the gastric glands. I, therefore, have made the following modification of technique, which does not materially interfere with secretion, and which permits of an accurate preliminary fluoroscopic outline of the fasting stomach followed by a modified Ewald test meal which permits of a brief fluoroscopic study of the pyloric region before the barium mixes with the supernated injusta. The patient is placed behind the fluoroscopic screen and given to drink two ounces of water in which is stirred up ten to twelve grams of barium, as suggested by Dr. Case. This is readily seen entering the stomach and gravitating to the lower outline of the magenblase, its further behavior depending upon the tonicity of the musculature of the potential tube which constitutes the greater part of the average vertical stomach. If this musculature has a normal tonicity, the barium will be seen lingering for a brief time in a semicircular or inverted pyramidal form. It will then be seen to more or less rapidly canalize the stomach and form a dark streak for a variable distance and direction, depending upon the location of the pylorus. It then makes a sudden turn to the right, usually not very far from the level of the umbilicus and comes to rest as a more or less transverse curved shadow with its concavity upwards from one to two cm. in diameter and five or six cm. long, its right extremity not infrequently being on a level with or even exactly coinciding with the umbilicus. This is, of course, the J shaped or cow horn shaped terminus of the average vertical stomach and corresponds with the so-called pyloric antrum, which really is not an anatomical entity, but simply the distal end of the stomach segmented by muscular contraction. If there is hypertonicity, the barium will linger longer at the bottom of the magenblase, or if there is hypotonicity no perceptible pause may be seen, the barium at once being seen at the pyloric end, its pathway having been marked by a somewhat broader and fainter -shadow. At this point palpation of the abdomen with a Holzknecht spoon behind the screen will enable one to identify the relationship of any tender pressure points, if such exist, to the various parts of the stomach, the precise topography and position of which can also be accurately noted and recorded on tracing paper if so desired.

Before removing the patient from behind the screen, he is made to drink a test meal composed of one and one-half ounces of cream of wheat boiled in a sufficient amount of water so that it will measure ten ounces when cooked. The movement of the barium in the pyloric antrum shows the amount of depression from the weight of the test meal which gives some information regarding the mobility and tonicity of the stomach and its supports. Pyloric function, especially patency and peristalsis, can be further studied at this point if the symptoms suggest that this is necessary or if not, the test meal may then be treated in the ordinary manner either by its fractional removal through an Einhorn duodenal tube which has been swallowed with the test meal or by the ordinary stomach tube at the close of the conventional one hour

The character of the test meal is a matter of some consequence with reference to the facility with which the gastric contents can be drawn through the perforated bulb of the Einhorn duodenal tube, in case this is used. The cream of wheat meal above indicated seems to meet the requirements better than such material as shredded wheat, bread, etc. The progress of digestion both as to acidity and chymification, can be observed at suitable intervals. I have found that if about 5 c.c. are removed every 15 minutes and submitted to routine analysis that not only the conventional results at the end of the hour are determined, but the rapidity of gastric digestion can be ascertained at any time during the hour. It is also perfectly obvious that the clinical observation can be extended beyond the hour in cases of slow digestion where such observations seems to be worth while. Some information in regard to motility is also obtained in this way, although a larger amount and possibly a different kind of meal is required to develop the actual motor power of the stomach. Such a preliminary investigation as this which is purely incidental to the general scheme of the clinical study will at least enable one to determine whether further roentgenologic and chemical studies will be necessary or not. The fluoroscopic study has at least been carried quite as far as the chemical, and nothing further will be needed along either line in the average case.

There is always great danger of exalting any important laboratory procedure such as a serologic or X-ray finding to the dignity of a diagnosis. The information which these methods give is important and should not, by any means, be neglected, but the general aspects of the case, as determined by a painstaking history, physical examination (of which the fluoroscopic examination is simply a part) and especially including generaly appearance, occupation, environment and other routine clinical data are even more important and must enter to a corresponding degree into the final judgment of the case.

Where further information is desired, other methods, such as, the fluoroscopic study of motility with a larger test meal and larger quantity of barium can be used, but the very simple routine above outlined will in a large proportion of cases furnish all the information needed with very little expenditure of time.

It has been found especially useful in a group of cases which come to the office of the consultant and in which some X-ray data seemed desirable. A very few minutes spent in the X-ray room adjoining the consultation room enables the diagnostician to either get all the information required for a rapid diagnosis, or, in the few cases in which this cannot be done, advise further study along the lines indicated.

If the widest possible service is to be rendered by X-ray examinations in routine diagnosis, something besides cumbersome and expensive serial pictures will be required. The fluoroscope is the alternative and will probably in the very large majority of cases meet all requirements. The specialist in diagnosis should have it at his fingers' ends and make it one of the first rather than one of the last resorts. By sweeping the screen over chest and abdomen with the slight addition suggested in this paper as a procedure, which either may be preliminary or final, he can in a very few minutes time get some information that he cannot get without an X-ray examination and, in addition, get a great deal of information more accurately than in any other manner in regard to the general physical condition of the contents of both of these cavities.

SUMMARY.

By the fluoroscopic observation of about one-third of an ounce of barium stirred up in two ounces of water in its passage to the pylorus, we can get an absolutely accurate outline of the position of the fasting stomach in a perfectly quiescent state without distortion by the weight of a heavy meal. Muscular tonicity is well indicated by the time that the barium lingers in the magenblase and the rapidity of the canalization of the stomach. The coincidence of a point of tenderness on pressure, or a palpable tumor with the pylorus or other part of the stomach can be instantly determined. If chemical study of gastric secretion is desired, large quantities of barium cannot be used as they inhibit the secretion of hydrochloric acid. The quantity above indicated only slightly interfere with secretion and the turbidity caused by mixing of the barium with the test meal does not substantially interfere with the titration of acidity. The delicacy of the Ufflemann test for lactic acid is diminished by about one-half. The test for occult blood is not perceptibly interfered with. In a very large proportion of cases, this technique with such modifications as the individual case, or the judgment of the clinician may indicate is quite sufficient for the purposes of diagnosis and will either exclude serious organic lesions or taken in conjunction with other available data, especially the anamanesis, indicate further study along the proper lines of clinical investigation. It is only by the routine use of fluoroscopy that the diagnostic aid of roentgenology can obtain that widespread use so desirable in the field of internal diagnosis.

DISCUSSIONS.

Dr. John C. Hemmeter, Baltimore: Mr. President and Members of the Association: Dr. McCaskey has thrown out a suggestion on which he did not lay the most important emphasis in his paper, because he emphasized the relative value of the roentgenogram and the fluoroscopic method. Incidentally, he said, that barium reduces the secretions of hydrochloric acid. Dr. McCaskey makes a therapeutic suggestion which is of importance. I should like to ask how he ascertained this? What was his method? Did he use barium carbonate?

Dr. McCaskey: Barium sulphate.

Dr. Hemmeter: I should like to know how Dr. McCaskey ascertained that the barium sulphate reduced the secretion with hydrochloric acid. I should also like to know how he proceeded. Then the suggestion that the barium reduces the amount of hydrochloric acid is one that you do not find in literature. Any insoluble substance covering the gastric mucosa will reduce the amount secreted. I know it is the case with common kaolin or albumin silicate.

Dr. S. J. Meltzer, New York City: May I avail myself of the opportunity to put a question, not so much to the reader of the paper alone, as to any one who had some experience with the subject? The question is, what effect does barium exert upon the velocity of intestinal peristalsis? My own experience in animals is that barium hastens peristalsis. This question in fact is not the essential point which I wish to bring out. If you permit me, Mr. President, I shall try to smuggle in here a remark which I intended to bring out. If Dr. Carlson were here and read his announced paper—a remark which has been present in my mind for many years. Ever since Cannon began to study the character of the physiological peristalsis by means of X-ray and bismuth on cats a simple question occurred to me. Any one who has been in medical practice knows that bismuth is used in the treatment of diarrhea. I remember the time when 0.3 gms. was given for this purpose. Later the dose was increased to one gram every four hours. It hardly ever failed to stop the diarrhea and the abnormal peristalsis. Now let us look at the foundation of the entire study. I do not mean the study of *pathological* condition by means of X-ray, I mean the *physiological* conclusions drawn from such methods. In these experiments as a rule a cat of three kilos received five grams of bismuth. Now let us put in the place of the cat a man weighing seventy-five kilograms; he would have to receive in proportion one hundred and twenty-five grams in a single dose. What effect would such a dose have on the normal peristalsis of such a man? Will his bowels move sooner than once a week? It seemed to me quite probable that what was seen by the X-ray was the movement of the bismuth *within* the gut and not of the actual physiological movements of the *gut itself*. Is it not possible that it was for this reason that Cannon introduced a physiological form of peristalsis which he termed "segmenting?" With that amount of bismuth in its lumen the gut could make only small motions which appeared in the X-ray picture as peristaltic segmentation. I put this question to all of you who, I am sure, know a great deal more of the literature on that subject. To me it seemed all these years that it was not warranted to draw such conclusions from the facts which Cannon observed. I may be wrong in my analysis; I wish Dr. Carlson were here to discuss the subject. My question may have been answered before without my knowing it. You will pardon me for smuggling in my remark; if barium *hastens* peristalsis it would also not be a proper means of studying the cause of normal peristalsis. I wish to emphasize again that my remarks have no reference to the statements made with regard to abnormal peristalsis.

Dr. George R. Lockwood, New York: It seems to me, after some years of experience, that Dr. McCaskey's method of using barium water and watching it go down with the fluoroscope is a good one. I can thoroughly endorse every word in his paper, and can speak most enthusiastically for the results of fluoroscopy as compared with radiographic work in all cases in which fine demonstration of the object is not called for. I have only one suggestion to make regarding the paper. It seems to me that the fluoroscopic room should be jet-black, like the photometric room of gas analysis; and that we should sit in the dark room for half an hour, and certainly not less than twenty minutes. I can see nothing in the fluoroscope within less than that time. In going from a bright light to this room, one loses the most essential parts of the picture.

Dr. Jacob Kaufmann, New York City: I should like to answer Dr. Meltzer's question, considering the older literature on the effect of bismuth—and other meals on gastric peristalsis. A very extensive discussion followed an article by Hiller,* who tried to show that bismuth and other meals influence the tone and motility of the stomach to such an extent that artificial conditions are provoked which do not permit to draw conclusions from X-ray pictures considering physiologic conditions. The discussion was carried on for several years. Hiller maintained that X-ray meals actually bring about contractions of the stomach, which give it an entirely different configuration from that which the stomach has in its normal condition, so that deductions drawn exclusively from X-ray examinations are bound to be erroneous, if interpreted as being physiological.

Dr. F. W. White, Boston Mass.: Regarding Dr. McCaskey's paper, I feel as he does with reference to the value of the fluoroscope, and the lack of necessity for plates in a good many cases. In some cases sharp detail is necessary, however; and then we must have plates taken. The method recommended by Dr. McCaskey is used pretty generally over the country. The Mayos do the same thing; and we do, in the Boston hospitals. It is our routine method of handling a lot of patients to get the best results in a short time.

My feeling about the combined meal is that I should rather not use it. I let the patient take his test breakfast at home and then come to me. At the end of fifty minutes, I take out the residue and, a few minutes afterwards, give a barium meal and make the fluoroscopic examination. In this way, we get good results, without mixing factors. It takes only five minutes to get rid of the test breakfast, and then we go into the fluoroscopic room to look at the patient's stomach. The whole thing is over in a very short time.

DISCUSSIONS.

Dr. F. W. White, Boston: I should like to say something about diagnosis and treatment. In looking at Roentgen plates of diverticula, one must be careful not to confuse them with small haustrae of the bowel.

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*Cf. Arch f. Virdaumozrkr. Bd. XVI.

Regarding the treatment, I use an even simpler method, namely, a large dose, several ounces, of barium sulphate or bismuth subcarbonate to fill the diverticula and prevent other material from stagnating in, and irritating them. I got my idea from the Roentgen examination, by finding how much some patients were relieved by the usual large dose of barium sulphate by mouth.

DR. MCCASKEY, closing: Dr. Hemmeter asked about the combination of the barium with hydrochloric acid. The method I use is to take a watery solution of hydrochloric acid, which by titrations show 50°, and then add barium and titrate again. I find no change. It does not interfere with the reaction. I then mix a protein and egg albumin and put it into the incubator over night, and no change occurs. It is still 50°. Therefore, I conclude that the only thing that will reduce the amount of hydrochloric acid deficiency in the action of the acid glands. That is all there is to it.

I might say one or two things additional. The barium does not interfere with the blood test. I could detect no difference. It interferes a little with the lactic-acid test, reducing it about one-half.

Regarding the peristalsis question raised by Dr. Meltzer, the thought occurred to me that if it inhibit secretion, it might inhibit peristalsis. I have some clinical studies on this point under way, but have no personal experience and would like Dr. Meltzer to tell us if he knows what the literature is on the question.

With regard to the adjustments of the eyes for fluoroscopic examinations in the dark, I do not have much trouble. I think that five minutes is long enough for this in the way I manage it. If you go from a bright room into the dark room, fifteen minutes is necessary; but when I am in my consulting room and expect to make a fluoroscopic examination, I put on a pair of goggles and darken the room a little. My clinical clerk goes on taking the history while my eyes are being accustomed to the lessened amount of light; and when I go into the fluoroscopic room, I am able to see all I ever could see in less than five minutes. My eyes are very well prepared. I think that the longer I stay, within certain limits, the more I can see; but I do not think that it is necessary to remain twenty to thirty minutes, especially with the preparation in the consultation room before I go in.

Then about the advantage of the combination meal, I do not know. It seems to me that in the one hour's time I can get more information than I can otherwise get in twice that time. Of course, I have not gone far. In cases in which more study is needed, it is necessary to give more time; but this is simply intended to give me preliminary information regarding tonicity, position, etc.

Outside of the lactic-acid interference, which is not very important, it does not in any way change the result, so far as I can see. The barium turbidity does not interfere with the blood test. I have not tried the proteid test. My idea was to help popularize the use of the fluoroscope as a routine measure of observation.

SOME OBSERVATIONS ON GASTRIC AND DUODENAL ULCER.

By J. C. Johnson, M. D.

Several years ago we published a review of forty cases of ulcer. In this review we referred especially to the frequent association of ulcer with hyperchlorhydria, hypersecretion and gastrosucchorea, and endeavored to draw some conclusions as to the relation between them all. It appeared that there was no symptom common to these disorders always present, and no condition in any which might be considered pathognomonic of ulcer. But we believed then, and still are of the opinion, that ulcer is no more an entity than is any other disease of the stomach or duodenum, and that even as a clinical entity its existence may be questioned. However, before an intelligent idea can be formed as to the relation between two or more concurrent diseases we must first have definite knowledge of each separate disease, and must also be sure that what is classed as disease is something more than a phase or an epoch of disease.

In any event a study of ulcer cannot be comprehensive which does not take into account each distinct process which may predispose to it or which may be present with it.

Experimental production of ulcer has taught us much of value. But experiment cannot prove everything about anything. Art cannot create the conditions which develop as a sequence of orderly physiological perversion.

Surgery has given us a closer view of the morbid anatomy of ulcer and of the deformities which often follow it. But surgery can never determine the cause of, nor depict the tissue change incident to its development. Fortunately there are other ways of knowing things than by seeing them, and other ways of finding out than by probing. The very problems which call for radical action often escape the eye and are discernible only to reason and common sense.

The truth about ulcer has its foundation in the stomach and duodenum themselves, and it can only be expressed in terms of pathology most intimately related to their functions. Upon this presumption we have tabulated other cases, fifty in number,* in order to enlarge our data and add something to our knowledge of this very common and very serious affection.

It may be noticed that six or twelve per cent. of these patients were under twenty years of age.

Seventeen, or thirty-four per cent., between twenty and thirty.

Fourteen, or twenty-eight per cent., between thirty and forty.

Ten, or twenty per cent., between forty and fifty.

Three, or six per cent., between fifty and sixty.

Three, or six per cent., between sixty and seventy.

Thirty, or sixty per cent., were males. Twenty, or forty per cent., were females.

Various occupations were represented.

The history of previous indigestion varied from six months to a lifetime.

The highest Hgb. was 88. The lowest 105.

In fourteen, or twenty-eight per cent., the greater curvature of the stomach was below the umbilicus—in some instances reaching almost to the pubis.

In eighteen, or thirty-six per cent., the greater curvature extended to the umbilicus.

In eighteen, or thirty-six per cent., the position and size of the stomach were apparently normal.

Thirty-nine, or seventy-eight per cent., were constipated.

In nine, or eighteen per cent., there was a daily stool.

Two, or four per cent., had diarrhea.

Local tenderness was present in thirty-nine, or seventy-eight per cent. In thirty, or sixty per cent., the tenderness or soreness was referred to the epigastric region. In four, or eight per cent., to the right hypochondrium. In two, or four per cent., to the umbilicus.

In two, or four per cent., to the left hypochondrium. In one, or two per cent., to the Xiphoid angle.

Next in frequency to soreness was pain—present in thirty, or sixty-eight per cent. In twenty-three, or forty-six per cent., it was referred to the epigastrium. In three, or six per cent., to the pectoral region. In two, or four per cent., to the right hypochondrium, episgastrium and left hypochondrium. In two, or four per cent., to the left iliac. In two, or four per cent., right iliac. In one each, or two per cent., respectively, to the left hypochondrium, Xiphoid angle, umbilical regions.

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*Of these, 28 were duodenal, 16 gastric, and 6 pyloric.

Nausea was present in fifteen, or thirty per cent. Vomiting in twelve, or twenty-four per cent. Hemorrhage was rare. Occult feces was positive in six, or twelve per cent.

In twenty-one, or forty-two per cent., there was evidence of family tendency to gastric-intestinal disorder.

Four were members of the same family—including father, mother and two sons.

In twenty-four, or forty-eight per cent., the history of family tendency was negative. In the remaining five, or ten per cent., the history was indefinite.

Six, or twelve per cent., had appendectomy, prior to examination, one or more as long as seven years.

In one the gall bladder had been drained. In one tonsils had been removed.

The extent of the examination and the methods used in each case are suggested by the table. Besides, no effort was spared to discover everything in the history and general condition of the patient that could have any bearing on the ulcer. Only that which we have considered pertinent is included in this report.

We do not agree with those who say that a diagnosis of ulcer can always be made by telephone or by mail. Ulcer is sometimes present when it is not suspected, and sometimes suspected when it is absent. We are not sure that the most careful surgeon may not overlook an ulcer when he is looking for it. All ulcers are not the same in size, site or period of development. Positive diagnosis of ulcer can be based only upon finding one.

There are some who say that the stomach tube, the laboratory and other means employed by the internist are unreliable and of little value. The ground upon which these observers stand is so remote from this particular field of scientific progress, they hear no sound from without, but the echo of their own voices. They see nothing beyond the horizon of their own sphere. They are so firmly fixed in their opinions by the centrifugal nature of their thoughts no ideas but their own can have accession, persuasion or cohesive power.

We would pass this point unnoticed did it not quicken us to reaffirm our faith in those verities which they deny without first having knowledge of them. The science of medicine is too deep and too broad for any man however tall to stand upon its bottom anywhere and reach from one extereme to the other.

Unless one has had a wide experience in certain lines of work by what authority may he speak so assuredly of what can and cannot be accomplished by other methods than his own. By what inspiration can he know what it has required years of arduous labor for others to attain.

It is inconsistent to say the least of it to speak of the danger of cancer from ulcer as a base and of the need of early diagnosis, and at the same time to decry the means we have without supplying something better, or even suggesting something just as good.

It is not easy to distinguish between gastric and duodenal ulcer in every case. Pain is not invariably present in either, nor does it always occur at a time so definite that a diagnosis may be based upon this symptom alone. The point of local tenderness in both also varies, as does the pain, according to the position of the stomach and duodenum, and the character and extent of functional disturbance. Even with malignant ulcer far advanced there may be an absence of both pain and soreness.

Stenosis alone is not diagnostic of ulcer, neither does it always enable us to locate one when it exists. Only by determination of all the conditions present can the necessary knowledge and significance of ulcer be obtained, and the needs of the patient be intelligently supplied. There is no symptom so slight, or seemingly without value which may with reason be disregarded.

For location of ulcer there is nothing more reliable than the Einhorn Thread Test, and for detection when in doubt it is of equal service.

The X-ray will not picture an ulcer in its early stages or before a crater has developed. On the other hand it may show a deformity resembling that due to ulcer, and yet the trouble may be one in which the stomach is not primarily involved.

The table shows that in 94 per cent. the Thread was positive, whereas the X-ray when used was positive in 68 per cent.

The Thread may not be positive for ulcer on the greater curvature towards the splenic end, or where the ulcer has healed, or is healing.

There is no theory of ulcer convertible into fact which detaches its formation from the histological and biological forces of normal tissue change.

We have before made occasion to express the belief that the primary causes of disease are overwork, underwork, injury and infection. If this classification is correct, it narrows the field of our present investigation and simplifies the result.

Fatigue of any organ predisposes to disease, even if it may not constitute a condition which within itself is technically disease. Certainly it is often a controlling factor in the conduct of disease. The predisposition created by fatigue rests essentially in impairment of nutrition. But it is misleading and confusing to regard nutrition as a process. It is not a process, but a condition the result of many processes—and hence cannot be the cause primarily of any other process.

Fatigue of the stomach may be the result of effort to overcome resistance in some other organ to which it is intimately related, as the duodenum, liver or heart. It may follow excessive functioning stimulated or required by errors in diet. It may also develop from continued exercise within normal limits, but with insufficient material for repair. The duodenum may suffer in a similar way from similar causes.

It is significant that in 42 per cent. of the patients there was a family history of gastro-intestinal disorder, and that four of the patients were father, mother and two sons. This suggests that a certain habit of metabolism, or state of nutrition predisposes to ulcer. Yet in others this history is lacking or indefinite. Whether ulcer be local or constitutional in origin, somatic metabolism as a causative factor however potent and essential must be more or less indirect. And even if essential, and the primary importance of gastric and duodenal function is denied, it remains to be explained why these organs and not others are selected for the local manifestation of the general disease.

Traumatism predisposes to actual disease, but unless the continuity of the tissue is disturbed to the extent that healing is impossible, disease does not follow. Even when the function of the tissue has been impaired before the injury is received, it resumes its former tone and efficiency when the disorganization from the injury has been overcome unless infection supervenes. When infection is added to traumatism or occurs independently the morphological changes which follow do not represent all the characteristics of necrosis and reformation found in classical ulcer. Furthermore, before infection can occur, can produce, or become active in a morbid process there must pre-exist histological and functional condition of the tissues not normally a part or product of organic life, unless there is possessed by some an immunity which is lacking in others. So far, we have nothing upon which to build a theory of immunity from ulcer.

The presence of Hcl. in the stomach is not favorable to pathogenic bacteria. Even when Hcl. is absent the protective power of normal gastric protoplasm may remain and repel invasion by microorganisms. This is often seen in achylia gastrica. If it were a fact that the protoplasm cannot be normal with Hcl. absent it would only lend support to our contention that the majority of gastro-intestinal diseases are primary in their essential nature and that ulcer as a feature is not the result of any single cause, but of an association of causes. But in a majority of ulcer patients there is an excess of Hcl., which leaves no room for argument that general impairment of gastric protoplasm is either a singular cause or a result of ulcer. Neither does it add anything to the theory of infection. Especially untenable is the idea advanced by some that ulcer is not only due primarily to infection, but that the appendix is the source of the infection. We found ulcer in 7 or 14 per cent. of the cases months or years after the appendix had been removed, and after other so-called surgical lesions had been repaired. It is evident that the ulcer in these cases was independent of the appendix or was overlooked at the time of the operation. In either event the need of more comprehensive knowledge and accurate diagnosis of gastro-intestinal diseases is hereby clearly demonstrated.

Much of the fallacy in modern teaching has its source in efforts to build the whole truth out of half the substance. Ulcer is not a ready made, hard and dried disease, but a gradual outgrowth of successive perversions in nutritive and functional activity, and only by appreciation of this fact and by proper employment of the means which it supplies can the best results from treatment be obtained.

Internists know that ulcers do get well without the knife, and that after many years these patients have all the appearance and comfort of perfect health. On the other hand it is true that there is no successful treatment for some ulcers or for the complications which they bring about except by operation. But the ideal operation for ulcer will be selected only when the surgeon keeps before him the complex relation of the alimentary functions and the more complex forces which make possible and maintain these functions normally, and brings all this to bear in interpreting the particular requirements in the field of operation. A true surgeon always does this. His task is made easier and his burden lighter if aided by the previous observations and present counsel of the internist. Their combined intelligence is not too much for any case.

For lack of these considerations or for lack of opportunity to apply them, operations often fail not only to restore to normal the parts primarily affected, but also fail to preserve that measure of compensatory and reciprocal action which has remained despite the disease.

Rec. No.	Age	Sex	Occup.	History of Indigest'n	Hgb.	B.P.	Pos. Stom.	LocalTen-derness	Pain	Naus.	Vomit	Stools	Occult Feces	Test Meal	Thrd.	X-Ray
46	34	F.	House	6 mos.	60	115	To U.	Pit	Neg.	Neg.	Neg.	C.	Neg.	ʒII 46.64	Pos.	......
39	46	M.	....	15 mos.	75	100	To U.	Neg.	Neg.	Pos.	Neg.	Lax.	Neg.	ʒI 30.52	Pos.	......
93	28	M.	Farm	15 yrs.	80	120	To U.	Pit	Neg.	Pos.	Pos.	C.	Neg.	ʒV 80.100	Pos.	Neg.
88-A	44	M.	SalesT.	10 yrs.	80	105	Sub.U.	Epig.	X-Phoid	Neg.	Frcd.	Reg.	Neg.	ʒVI 70.88	Pos.	Pos.
487	23	M.	Bnk.	9 mos.	80	125	Situ.	Pit	Pit	Neg.	Neg.	C.	......	ʒII 66.86	Pos.	Neg.
342	28	M.	Clerk	Months	75	130	Hr.Gl.	Pit	Pit	Neg.	Neg.	C.	Neg.	ʒVI 46.68	Pos.	......
194	23	M.	....	1½ yrs.	80	115	Atonic	Pit	Neg.	Neg.	Neg.	D.	Neg.	ʒIV 80.114	Pos.	......
179	19	M.	Clerk	1 yr.	80	120	Situ.	Neg.	Neg.	Neg.	Eruct.	C.	Neg.	ʒIV 42.64	Pos.	......
539	52	F.	House	Years	75	120	Sub.U.	Left U.	Neg.	Neg.	Neg.	R.	Neg.	ʒss 64.94	Pos.	......
721	67	M.	Mill	Years	80	130	Sub.U.	Pit	Pit	Neg.	Neg.	C.	Neg.	ʒVI 74.100	Pos.	......
88-B	18	M.	Clerk	5 yrs.	83	100	Situ.	Pit	Pit	Neg.	Neg.	C pl.	Neg.	ʒVI 108.132	Pos.	......
44	36	F.	House	Years	75	160	Sub.U.	Neg.	Neg.	Neg.	Neg.	R.	Neg.	ʒss 14.32	Pos.	......
335	36	F.	House	?	75	120	Situ.	Pit	Pit	Neg.	Pos.	C.	Pos.	ʒIss 52.72	Pos.	Pos.
629	47	M.	Atty.	20 yrs.	.....	.....	Situ.	....	Pit	Neg.	Neg.	C.	Pos.	ʒX 58.70	Pos.	Pos.
479	30	M.	Sales	18 yrs.	80	110	Situ.	X	Pit	Neg.	Neg.	R.	Pos.	ʒXI 70.100	Pos.	Neg.
352	25	F.	House	5 yrs.	80	.....	Situ.	Pit	Pit	Neg.	Neg.	C.	Neg.	ʒIIss62.100	Pos.	......
533	45	F.	House	?	78	115	Situ.	Neg.	Neg.	Neg.	Neg.	C.	Neg.	ʒI 22.42	Pos.	Pos.
864	47	F.	House	? yrs.	.....	.....	Sub.U.	Pit	Pit	Neg.	......	C.	Neg.	ʒss 50.64	Pos.	......
108	36	M.	Bk.	12 yrs.	83	135	....	Pit	Neg.	Neg.	Neg.	R.	Neg.	ʒIX 42.66	Pos.	Pos.
462	40	M.	R.R.	7 yrs.	80	120	....	Pit	Pit	Pos.	Pos.	C.	Neg.	ʒII 72.100	Neg.	Pos.
367	31	F.	House	1 yr.	80	115	To U.	Pit	Pit	Pos.	Pos.	C.	Neg.	ʒVI 66.86	Pos.	......
385	65	F.	House	Years	70	195	Sub.U.	Neg.	Neg.	Pos.	Pos.	R.	Neg.	ʒIV 68.92	Pos.	Neg.
909	28	M.	Trns.	Yr.plus	78	130	Situ.	Pit	Pit	Pos.	Neg.	C.	Neg.	ʒII 36.48	Pos.	......
855	29	M.	Sale	6 mos.	80	116	To U.	Pit	Neg.	Neg.	Neg.	Reg.	Neg.	ʒIV 84.116	Pos.	......
850	19	M.	Farm	8 yrs.	80	120	To U.	Pit	L. pct.	Neg.	Neg.	C.	Neg.	ʒVI 78.104	Pos.	Pos.
37	28	M.	Plant	2 yrs.	80	115	To U.	Pit	Pit	Neg.	Neg.	C.	Pos.	ʒIX 72.92	Pos.	Pos.
892	24	M.	Sale	10 yrs.	80	120	To U.	Pit (R)	Pit (R)	Neg.	Neg.	C.	Neg.	ʒIV 74.96	Pos.	Pos.
287	48	M.	Dnt.	5 yrs.	.....	.....	Situ.	Neg.	Ret. R. & L.	Neg.	Neg.	C.	Neg.	ʒIV 86.104	Pos.	Pos.

Rec. No.	Age	Sex	Occup.	Previous History	Hgb.	B.P.	Pos. Stom.	Local tenderness	Pain	Naus.	Vom.	Stools	Occult Feces	Test Meal	Thrd.	X-Ray
659	24	F.	H.W.	Sev'l yrs.	80	115	Sub.U.	Pit	Left iliac	Pos.	Neg.	C.	Neg.	ʒIV 54.88	Pos.	Neg.
290	41	F.	H.W.	Sev'l yrs.	75	110	Sub.U.	Neg.								
							Pos.	Neg.	Lower epig.	Pos.	Pos.	C.	Neg.	ʒIV 46.72	Neg.	Pos.
869	41	M.	Farm	7 yrs.	80	130	To U.		Pit	Pos.	Pos.	C.	Neg.	ʒII 80.108	Pos.	Pos.
62	26	F.	S.M.	10 yrs.	80	110	Sub.U.	U.	Neg.	Neg.	Neg.	R.	Neg.	ʒI 62.78	Pos.	Neg.
326	35	M.	Atty.	Sev'l yrs.	88	135	U.	Pit	Pit	Neg.	Neg.	C.	Neg.	ʒIX 84.90	Pos.	......
514	21	F.	School	9 yrs.	.....	.....	Situ.?	Pit	Neg.	Neg.	Eruc.	C.	Neg.	ʒII 40.70	Pos.	......
505	32	M.	Merc.	8 yrs.	85	125	Above									
							U.	Pit	Lower pac.	Neg.	Neg.	C.	Neg.	ʒVI 56.80	Pos.	Pos.
593	45	M.	Merc.	15 yrs.	88	135	To U.	R. cost	R. iliac	Pos.	Neg.	C.	Neg.	ʒIV 86.104	Pos.	Pos.
238	21	M.	Clerk	8 mos.	80	125	Situ.	Neg.	Stom.	Neg.	Neg.	C.	Neg.	ʒIV 66.100	Pos.	Pos.
540	36	M.	Farm	15 yrs.	70	145	To U.	R.I.&Pit	Pit	Neg.	Neg.	C.	Neg.	ʒII 62.116	Pos.	......
375	23	M.	L'wy'r	2 yrs.	85	115	To U.	Neg.	Pit	Neg.	Neg.	C.	Neg.	ʒVI 42.78	Pos.	Pos.
317	34	F.	House	Years	70	90	Sub.U.	Pit	Pit	Pos.	Neg.	C.	Neg.	ʒVI 54.88	Pos.	Pos.
215	26	M.	....	Sev'l yrs.	80	100	Sub.U.	Pit	Neg.	Neg.	Neg.	C.	Pos.	ʒIIss40.72	Pos.	Pos.
916	28	M.	Drug	8 yrs.	80	120	To U.	Pit	Pit	Neg.	Neg.	C.	Neg.	ʒVI 64.80	Pos.	......
270	44	M.	Farm	10 yrs.	78	120	Situ.	Left cost.	Left cost.	Neg.	Neg.	C.	Neg.	ʒIII 80.100	Pos.	Neg.
263	37	M.	Farm	Since												
				child	78	105	Sub.U.	Pit	Neg.	Pos.	Neg.	C.	Neg.	ʒIV 78.98	Pos.	Neg.
167-A	18	M.	Neg.	5 yrs.	80	90	To U.	Left pit	Right pit	Neg.	Pos.	C.	......	ʒIV 49.65	Pos.	Neg.
389	50	F.	House	Sev'l yrs.	75	115	Silic.	Pit	Pit	Pos.	Neg.	C.	Neg.	ʒI 44.66	Pos.	......
210	38	F.	....	2 yrs.	75	140	To U.	Pit	Neg.	Neg.	Neg.	C.	Neg.	ʒIII 50.70	Pos.	......
615	52	F.	House	23 yrs.	75	145	To U.	Pit	Side	Neg.	Neg.	C.	Neg.	ʒIV 70.100	Pos.	Pos.
521	31	F.	House	Years	80	130	To U.	Pit	Neg.	Pos.	Pos.	C.	Neg.	ʒI 62.94	Pos.	......
167-B	34	F.	House	6 yrs.	75	130	To U.	Stom.	Umb.	Pos.	Neg.	R.	Neg.	ʒI 26.92	Pos.	Neg.

ROENTGENOLOGICAL STUDIES IN THE HEALING OF GASTRIC AND DUODENAL ULCER.

By Walter W. Hamburger,*

CHICAGO.

(From the Medical Series of Michael Reese and Cook County Hospitals, Chicago.)

It is a common experience, that the interpretation of *cure* of chronic gastric ulcer is difficult, because of the spontaneous remissions and exacerbations of symptoms, so frequent in the clinical history of these patients. The need of more definite and objective evidence of cure has been apparent for some time. In the attempt to secure such evidence we have been interested during the past two years in studying these patients by means of repeated roentgenological examinations before, during and following medical treatment. Our method is as follows:

First.—In addition to the regular routine of history taking, physical examination, test meal examination, etc., the patient is subjected to a thorough preliminary roentgenological study, including the registering of from six to a dozen plates, taken at intervals after the barium meal.

Second.—In the event that positive roentgenographic evidence of ulcer is thus obtained the patient is put under medical treatment (the details of which do not concern us here) for from three to six weeks. Within this time and before discharge from the hospital the patient is again thoroughly studied roentgenologically, including the making of plates.

Third.—The patient is asked to report back to the hospital at least every three months for a repetition of the roentgenographic studies.

In other words, roentgenological examinations are made before, during and after medical treatment, as often as every three months, for a period of one to two years or longer. These studies serve as a guide to the healing process, and as a contrast with the findings before treatment.

Thus far we have studied in this fashion eleven cases, of which seven are lesser curvature cases, and four are duodenal.

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*Dr. Hamburger's illustrations may be found in the *American Journal of the Medical Sciences*, February edition (1918).

In this paper I shall refer very briefly to the results of this study.

I. Lesser Curvature Ulcer.

Haudek,[*] in 1910, described his classical "*Nischen Symptom,*" which was the demonstration roentgenologically of a small round projection ("Ründlicher Vorsprung") along the lesser curvature of the stomach. Haudek believed that this projection or pocket represented the accumulation of bismuth or barium in the crater of a penetrating ulcer. In certain cases the pocket or crater of bismuth was surmounted by a small air bubble which could be observed in the plate.

Haudek's observations and conclusions have been confirmed by many workers both in this country and abroad. During the course of our work we have had abundant opportunity to examine such stomachs either at the operating table or at autopsy, and have likewise been assured of the correctness of Haudek's work.

Cole,[**] in 1915, amplified Haudek's work, reporting a series of indurated gastric ulcers of the lesser curvature—with or without the niche—detected at a far earlier stage. Cole's method, which he describes, is that of serial roentgenography. Many of our plates, particularly those taken after medical treatment, strikingly resemble those of Cole's indurated ulcers.

Faulhaber,[†] in 1912, emphasized the importance of the spastic hour-glass stomach, either in conjunction with the Haudek niche or alone, as evidence of callous ulcer. While undoubtedly such hour-glass contractions occur, in our experience they have been quite uncommon—while the lesser curvature projection has been quite frequent. In one case with a very pronounced hour-glass and a lesser curvature niche the operation failed to reveal an an ulcer. This case will be referred to later in the discussion of the dangers of misinterpretation in this method of examination.

The positive identification of the niche to be in fact the ulcer crater may be seen in figures 1, 2 and 2a from Cases I, II and III.

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[*] M. Haudek, Die Roentgendiagnose des Kallösen Magengeschwüres und ihre Bedeutung. Münchener Med. Wochenschr. 1910, S. 2463.

[**] Lewis Gregory Cole. Roentgen Diagnosis of Indurated Gastric Ulcer. *Am. Jour. of Roentgenology*, November, 1915.

[†] Roentgendiagnostic der Magenkrankheiten von Dr. M. Faulhaber, Sammlung Zwangloser Abhandlungen aus dem Gebiete der Verdauungs und Stoffwechsel Krankheiten, IV Band, Heft 1, 1912.

The abstract of the history of the patient from whom figure 1 was taken is as follows:

CASE I. L. S., blacksmith, age 57. Michael Reese Hospital.

Complaint.—Pain in upper abdomen, loss of weight, recurring attacks every three to four months for the past two years. Pain sticking in character, one to two hours after meals. Two months ago had three attacks of hematemesis.

Physical Examination.—Slight loss of weight, tender point in mid-epigastrium, otherwise negative.

Test Meal Examination.—Continuous secretion, yeast cells, sarcinae, normal acidity.

Roentgenographic Findings.—"Lesser curvature contains a defect about two inches from the pylorus resembling that of a perforated ulcer." (Fig. 1.)

Marked gastric retention after six hours.

Operation.—Dr. L. A. Greensfelder: "On the posterior wall at the lesser curvature an easily palpable callous ulcer is found, approximately four centimeters from the pyloric sphincter (Fig. 1). The thickened callous area was about five centimeters in diameter and practically round. Pylorectomy, including resection of the ulcer, was performed."

In passing it is interesting to note that the location of the ulcer and its size corresponds very closely with the findings as obtained roentgenologically.

The history of patient from whom figure 2 was taken is as follows:

CASE II.—P. O., patient of the County Hospital with a typical recurring history of gastric ulcer for eight years.

Roentgenological Examination.—"Marked penetrating ulcer lesser curvature half way between cardia and pylorus." (Fig. 2.)

Operation.—Dr. Paul Oliver: "Deep callous ulcer found on lesser curvature in median portion of stomach. Ulcer resected. Gastro-enterostomy performed." (Fig. 2.)

Here also the close similarity between the X-ray findings and the ulcer may be noted.

Figure 2-a is taken from the following case:

CASE III.—J. R., car repairer, age 54. Cook County Hospital.

Present Complaint.—Began 14 months ago, consisting of epigastric pain 1½ hours after eating, nausea and vomiting.

X-ray.—"A rather large penetrating ulcer is seen on the lesser curvature of the stomach." (Fig. 2-a.)

Operation.—Dr. Paul Oliver: "Indurated ulcer on lesser curvature about 5 centimeters in diameter; some scar on serous surface with firm fibrous adhesions to the left lobe of the liver. Adhesions separated. Ulcer inverted by Lembert linen stitch. Posterior gastric jejunostomy."

While the ulcer itself was not resected and therefore could not be studied histologically, still the location and the findings agreed so exactly with the X-ray picture that there can be no doubt regarding the identification of the projection with the adherent callous ulcer.

These three representative cases I believe are convincing proof of Haudek's contention that the niche does in fact represent the crater of a callous ulcer. That these ulcers were in fact callous gastric ulcers and not carcinoma was proven by serial microscopic section, in none of which was any evidence of malignancy noted.

The permanent cure of the first two patients following operative removal of their ulcer, the first for nine months and the second for three years, is substantiating clinical proof of the nonmalignant character of their disease.

Accepting then, as definitely established that the barium projection represents the ulcer crater, let us turn to a consideration of the changes in morphology of the niche during and after medical treatment. In certain cases the depth and width of the projection gradually lessens, until, in the favorable cases, it has entirely disappeared, leaving a smooth, regular, lesser curvature outline. This disappearance of the niche can be followed accurately from week to week and month to month, and is usually accompanied with coincident improvement in the clinical findings, viz., disappearance of epigastric pain and distress; cessation of vomiting; disappearance of occult blood in stool and stomach contents; gain in weight, appetite, hemoglobin and general well being of the patient; gradual return to normal of gastric secretion and motility. Such ulcers may be said to be *healing,* and in the event that these objective and subjective changes continue satisfactorily, may, at the end of six to twenty-four months, be said to be *cured,* depending on the personal conservatism of the observer.

Certain other cases, however, do not progress so satisfactorily, either because of insufficient dietary care on the part of the patient, inaccuracy in the details or duration of medical management, or because of other underlying organic disease, such as carcinoma, syphilis, tuberculosis. In these ulcers the niche nar-

rows only temporarily, later resuming its former contour or even becoming deeper, wider and more irregular. Such patients demand either more accurate medical management, specific tubercular or luetic treatment, or *surgery*—or all three!

The following three cases have been chosen as representative of the changes in the niche before, during and after medical treatment:

Case IV.—L. B., male, 54 years of age. (History and permission to report this case kindly given by Dr. Jacobson. I am indebted to Dr. Rountree of the Presbyterian Hospital, for prints of the X-ray pictures.)

Complaint.—Patient has suffered from "indigestion" for 12 or 15 years, consisting of periodic exacerbations of severe colicky pain. At different times diagnosis of chronic gall bladder and appendix have been made.

Radiographic examination revealed a broad deep penetrating ulcer of the lesser curvature, midway between the cardia and the pylorus. (Fig. 3.)

After two weeks of medical treatment a second X-ray was made showing marked diminution in the depth and width of the lesser curvature pocket. (Fig. 4.) This coincided with parallel clinical improvements.

Four weeks later a third X-ray was made showing still more marked change in the lesser curvature. (Fig. 5.)

Three months after beginning of medical treatment a fourth X-ray was made showing complete disappearance of the lesser curvature projection. (Fig. 6.)

While the niche itself has disappeared one could see fluoroscopically that the normal peristaltic waves along the lesser curvature ceased in the area of the ulcer similar to the observation of Cole,* who calls this the "indurated area." In other words, apparently all that has happened is the filling up of the crater with organization tissue and possibly the transformation of a florid penetrating ulcer into either an indurated callous ulcer or an indurated scar on the lesser curvature. The absence of the normal peristaltic waves is conclusive evidence that complete restitution with absorption of all connective tissue has not occurred.

Case V.—A. T. H., a tea taster by occupation.

Complaint.—Late pains, vomiting, loss of weight, periodically, for five years.

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*Lewis Gregory Cole.—Roentgen Diagnosis of Indurated Gastric Ulcer.—*Am. Jour. of Roentgenology*, November, 1915.

Test Meals.—Subicidity, hypersecretion, motility normal.

X-ray.—Penetrating ulcer of the lesser curvature. (Fig. 7.)

Three months after treatment, during which time clinical progress was satisfactory, X-ray reveals slight convexity at site of original ulcer. (Fig. 8.)

Four months after treatment third X-ray practically normal lesser curvature contour. (Fig. 9.)

This patient has now remained free from all gastric distress for nearly two years since treatment was begun.

Case VI.—R. K.*, 52 years of age, a dye worker. (Service of Dr. J. C. Friedman.)

Present Complaint.—Epigastric pain for five years, vomiting, hematemesis.

X-ray.—Penetrating ulcer of the lesser curvature. (Fig. 10.)

Three months after treatment second roentgenological examination shows a lesser curvature defect still present, but a beginning narrowing and filling up of the ulcer crater. (Fig. 11.)

This patient has since disappeared from observation, but according to what scant information is at hand he is still having gastric symptoms.

The *results* of these studies bring up some rather interesting questions which may be discussed as follows:

(1) It is obvious that this method is of value in studying the changes occurring in callous ulcer during the healing process.

(2) The complete disappearance of the projection, *together with* parallel improvement in the patient's clinical condition is strong presumptive evidence of healing of ulcer.

(3) The complete disappearance of the pocket cannot be interpretated as complete normal restitution of the stomach wall, but probably is to be interpreted as simply the filling up of the ulcer crater, with probably the formation of an indurated callous ulcer or of a thickened scar. The failure of normal peristaltic waves to continue through the ulcer area is positive evidence of a remaining pathologic condition.

(4) The method is in no sense to be construed as an argument for or against medical treatment, but is of distinct value in the selection of cases for medical or surgical treatment. For in those cases, such as R. K. (Case VI) where the crater could not

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*This case, as well as cases Nos. VI, VIII and IX, have been reported in greater detail in the Medical Clinics of Chicago, Vol. 2, No. 3, pages 419 to 462.

be made to entirely disappear after prolonged medical treatment, such cases are undoubtedly worthy of surgical intervention.

(5) The method does not serve to rule out carcinoma. While in my own series I have yet to experience a lesser curvature ulcer which underwent malignant degeneration (or was cancer from the start and misdiagnosed), still the possibility of overlooking an early cancer must be granted, as emphasized by Cole, Case and others.

(6) Even though the ulcer crater has completely disappeared, resulting in the production of an indurated scar, permanent cure may not have been obtained. For it is possible that this scar, as in scars elsewhere in the body, remaining a *locus minoris resistentiae* may undergo secondary breaking down with the formation of fresh active ulcer.

(7) Extreme care and countless re-examinations in all positions, including the taking, if possible, of serial plates must be insisted upon to avoid the danger of overlooking the persistence of a small crater pocket. Even with such precautions, the danger of overlooking such small projections must be granted.

(8) Normal peristalsis may at times be mistaken for penetrating ulcer, as was forcibly impressed upon us by the following interesting experience (Case VII):

CASE VII.—(I am indebted to Dr. Kahlke for the details of this case, as well as permission to report same.)

Married woman, 26 years of age, symptoms for three years, consisting of late pains, hematemesis and bloody stools.

X-ray.—Hour-glass contraction of the stomach, indurated ulcer of the lesser curvature. (Fig. 12.)

Operation.—(Dr. Kahlke) "Following careful exploration of the entire stomach, anterior and posterior, cardia to pylorus, no evidence of ulcer could be found. Stomach was not opened. Appendix, gall bladder and ovaries, negative."

Cole has reported a similar case (Fig. 13). However, inasmuch as exploration of the stomach was not made *from within,* it is conceivable that a small penetrating ulcer was present, but was not diagnosed. That this is not an infrequent experience can be attested to by many surgeons and roentgenologists.

II. DUODENAL ULCER.

Cole,* in 1914, described his method of serial radiography for

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*Lewis Gregory Cole.—The Diagnosis of Post-Pyloric (Duodenal) Ulcer, *The Lancet,* May 2, 1914.

the diagnosis of duodenal ulcers based on the recognition of a "constant deformity of the cap or sphincter caused by the induration or cicatricial contraction surrounding the crater of an ulcer or resulting therefrom."

The method described above for the control of treatment of lesser curvature ulcer has been applied to the treatment of duodenal ulcer.

The following cases may be taken as representative of this type:

CASE VIII.—E. F. W**, merchant, 33 years of age.

Complaint.—Periodical for 12 years, distress after eating, black stools, vomiting of blood, loss of weight.

Test Meals.—Hyperacidity, normal motility.

X-ray.—Constant deformity of the duodenal bulb; motility less than six hours. Peristalsis active. (Fig. 14.)

Four and a half months after medical treatment second X-ray examination: The bulb was still defective but with possible lessened degree of contraction Peristalsis normal. No pylorospasm. Motility six hours. (Fig. 15.)

CASE IX.—I. E. S.,* attorney, 31 years of age.

Complaint.—Periodically for 18 years, fullness late after meals, belching, gnawing sensation at night.

Test Meals.—Hyperacidity, hypersecretion and delayed motility.

X-ray.—Peristalsis slow, considerable retention in six hours. Bulbus defective. (Fig. 16.)

One month after medical treatment: Normal peristalsis, motility four hours, bulbus still defective. (Fig. 17.)

This method applied to cases of duodenal ulcer, has not up to the present, been productive of as many positive findings as in the case of lesser curvature ulcer. Notwithstanding, it would appear that such repeated roentgenological examinations are of value and should be instituted as a routine in the medical treatment of all ulcers.

CONCLUSIONS.

(1) A method of roentgenological study of the healing of gastric and duodenal ulcer is described. This method consists of repeated roentgenological studies of ulcer patients before, during and at varying intervals after the institution of medical treatment.

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*This case has been reported in greater detail in the Medical Clinics of Chicago, Vol. 2, No. 3, pp. 419 to 462.

(2) This method is of value in the diagnosis, prognosis, control of medical treatment, and selection for surgical treatment of gastric and duodenal ulcer.

(3) The method is of value in studying the pathology of the healing process in both clinical and experimental ulcer. Thus far it is not of positive value in the differential diagnosis between ulcer and cancer, although in the future it may shed some light on the much discussed problem—the frequency of malignant degeneration of callous ulcer.

(4) In the use of the method the danger of mistaking normal peristalsis for penetrating ulcer and of the overlooking of the presence of a small ulcer, because of incomplete or insufficient examination must be borne in mind.

(5) These results are presented only as a preliminary report for the purpose of stimulating work along similar lines. It is in no sense to be construed as the final word on the subject, for each new case, as it is studied, brings its own message and suggests new viewpoints and problems to be interpreted, thus amplifying and possibly negating the conclusions reached in the preceding cases.*

DISCUSSIONS.

Dr. John A. Lichty, Pittsburgh, Pa.: I do not wish to discuss the papers, but should like to ask a few questions. First, I should like to inquire the extent of the pathology in Dr. Hamburger's cases that were operated on. In two cases that occurred during the past year in which I had similar X-ray presentations, I inferred that there was really a perforation with probably a localized peritonitis, sufficient adhesions being present to hold the ulcer from breaking through. In both cases, operation was strongly advised. One patient refused to have it undertaken, and perforation occurred within a week. The patient died, an autopsy was not obtained. In the second case, the ulcer was resected. What I thought was present was found. That is, the wall of the stomach was entirely destroyed. That being so, I can hardly see how we can temporize with these patients. If we are unable to tell by the X-ray how much of the stomach wall is destroyed, how extensive the lesion, I do not see how we can take a chance and wait without having an operation.

My second question refers to Dr. Johnson's paper. I am a strong advocate of the string test, but feel its shortcomings and wonder why I am so strong an advocate of it, after all. Dr. Johnson says that an ulcer at the greater curvature of the stomach may not show, whereas it may on the lesser curvature. I should like to know how he comes to this conclusion. What experimental evidence has he that will show that an ulcer on the greater curvature or towards the splenic end will not show a positive string test? I am of the same opinion as Dr. Johnson, but do not know why.

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*Most of the roentgenological examinations were made for me by Mr. Arendt, in charge of the X-ray department of the Michael Reese Hospital, to whom I herewith express appreciation for his assistance.

Dr. Charles G. Stockton, Buffalo, N. Y.: The deep inflammatory disturbances about the stomach following chronic perforating ulcer often make the surgical relief a serious operation. The first case of which I have any knowledge proved to be fatal. It was not under my immediate care; but I saw very soon afterwards a case of the same kind, and it seemed to me necessary that operation be done. It was a serious operation and there was a very stormy convalescence. The patient did poorly for a year afterward, and finally made a recovery or passed from under my attention when practically well. The second case was one similar to that shown by Dr. Hamburger, but with marked hour-glass contraction. The patient had been operated on six years before, under my advice, the ulcer being resected by Dr. Roswell Park. The cure lasted a year. I "cured" the patient five times afterwards, and then she came back with hour-glass stomach. I had "cured" her by the usual methods of treatment. This time, I thought it would be well to try duodenal feeding. The patient was so irritated by the tube, however, that it was vomited. Finally she was operated again, the operation consisting in breaking down very dense adhesions, the result of an intense perigastritis. Nothing was done with the ulcer. She did well afterwards. I had her again fluoroscoped and plates were made; the result showed that her condition was unchanged. The hour-glass contraction appeared to be more marked than before; but, strange to say, after three years, she reports herself entirely well.

The experience with the duodenal tube encouraged me to go farther. I have treated three cases in this way, two with hour-glass contraction and "niche" and one without "niche." These patients are reported well. I have seen two of them repeatedly, and they are as comfortable as the average person under a reasonably careful diet. Each shows hour-glass contraction, however, but not in so marked a degree as before.

Finally, I should like to call attention to the fact that in these admirable plates that Dr. Hamburger has shown, the hour-glass element is less in evidence than in my cases. I am surprised to find that so many chronic perforating ulcer cases are apparently without the hour-glass contraction. I want to say that I believe this sort of case, contrary to what has been said by the last speaker, is one that should receive very careful medical treatment; and that one should undertake surgery in such cases with a great deal of hesitation. I base that opinion on my own brief experience.

Dr. R. Walter Mills, St. Louis, Mo.: I think it is my duty to speak of two cases that we have had under observation in which the same thing occurred that Dr. Hamburger recited. One case has rare value, in that it was operated on and the diagnosis confirmed after the ulcer had healed. The first case was seen two years ago, and observed in intervals between attacks. A small ulcer crater was apparent on the lesser curvature. The case was observed again a year afterwards, when no crater was present. The patient went two years under medical treatment, without symptoms of any kind, on a rational, but pretty liberal diet. At the end of two years, symptoms again occurred; and the crater was again present, but distinctly smaller, it was perhaps half the size of the previous one. The other case was observed for two and one-half years. Four observations were made in all, and the crater which decreased gradually in size, studied. It was always present until the last examination, when it could scarcely be detected and probably would not have been recognized, had we not known before that it was present. I suspected that there was malignant degeneration about the ulcer site. The case was operated on, and the resection showed the ulcer practically healed. There was no crater, the site being filled with scar tissue. I think we should all appreciate it if Dr. Hamburger would tell us his experience with similar changes, if there be such, in duodenal ulcer.

I wish to say a word regarding Dr. McCaskey's paper. We have seen from Dr. Hamburger's paper, the extreme difficulty in differentiating ulcer evidence from peristaltic movements, artefacts and the like. While I am sure that Dr. McCaskey's procedure is a good one, it should be used with great care. The only way to differentiate these slight morphological changes and diagnose certain ulcers especially duodenal ulcers, is by the use of plates. Again, the early diagnosis of cancer is possible only by the combined plate and roentgenoscopic method. It seems that any procedure that tends to emphasize a short cut in cases in which the diagnosis is of such gravity and importance, should be distinctly understood as not a final diagnostic procedure.

JACOB KAUFMANN, M. D., New York: Dr. Hamburger attempts to show, as proof of the healing process of the ulcer that the X-ray picture of the Nische disappeared in some of his cases. On the other hand, I should not consider it proved that the ulcer did not heal if the X-ray picture of the Nische persisted, because ulcer may heal completely and the defect remain. That is what I would answer Dr. Lichty, if he considers the mere appearance in the picture of the defect. Let me cite an experience. A patient whom over a protracted period I had treated for gastric ulcer remained free of symptoms for fourteen years, when he returned again presenting severe ulcer symptoms, i. e., pains, pronounced gastric hypersecretion, vomiting, hemorrhage. The persistence of all these symptoms in spite of thorough medical treatment, led us to operate. The patient died and we had a post mortem. We found a healed ulcer, the size of a silver dollar, where the stomach wall had entirely disappeared, and the liver could be seen from the stomach side, with perfect scar tissue; yet all the symptoms of ulcer had reappeared, and we found in addition to the old defect a decided irritation and congestion of the gastric mucosa, showing numerous erosions and small ulcers of recent origin, which had produced all the symptoms that lead us to the opinion that these symptoms were associated with the old ulcer. Renewed symptoms of ulcer need not necessarily be associated with an ulcer previously diagnosed, treated and healed. The patient may develop a new one. The possibility of relieving a patient of disturbances which eventually lead to the formation of new ulcers or new disturbances in the old ulcer does not depend on methods of treatment, so far as surgical interference is concerned, or methods of diet and so on. It depends much more on whether we can remove the cause of the original disturbance of functions that led originally to the formation of the first ulcer. Here is the great difficulty in the treatment of gastric ulcer. That we have not, so far, cleared up the pathogenesis of ulcer at all. I think that is where our study ought to set in much more thoroughly. We must come to understand in each case what are the original disturbances of function, which, if they persist, lead to the formation of ulcers.

DR. ALLEN ARTHUR JONES, Buffalo, N. Y.: A woman who had no previous complaint of any gastric symptoms at all, developed irritative gastric symptoms, and a plate showed a typical crater, probably a little larger than the first Dr. Hamburger showed, on the upper part of the lesser curvature. After six weeks of medical treatment, the symptoms subsided, and another plate showed absolute disappearance of the crater. The patient has never had any recurrence of her gastric symptoms. Whether that was an acute ulcer or not, it is impossible to say. Of course, a person might have a chronic condition without symptoms. I think this was an acute ulcer, as shown by symptoms and by the roentgenogram, and that there was healing in six weeks, under rest and diet. I think this method of following up is of great value, as Dr. Hamburger has shown.

DR. MAX EINHORN, New York: The papers of both Dr. Johnson and Dr. Hamburger are of great interest. I fully agree with Dr. Johnson that there are several factors contributing to the production of ulcer. It is not one thing alone.

With regard to Dr. Hamburger's paper, I would say that it is very interesting to follow up the cure of these ulcers in the craters with the X-ray; I would mention some experiences of my own. There are two cases that I wish to speak of, which occurred quite recently. One was in a man with a dilated stomach and a history of ulcer of the pylorus. He had had ulcer for years. The X-ray showed obstruction of the pylorus and ulcer of the lesser curvature with crater. The patient had to be operated on, on account of the obstruction. He was very weak. The operation was done at the German Hospital and a gastroenterostomy established. The indurated areas at the lesser curvature was not touched. Patient made a complete recovery and remained well.

I will mention another case of equal importance. Here the X-ray was not of great aid, but the thread test showed ulcer of the duodenum. The clinical picture was that of cholecystitis. He had fever, a leucocyte count of 18,000, with 80 per cent. polynuclears and swelling of the liver and fever. The X-ray was not definite. It showed some adhesions around the liver and gall bladder. I examined the bile with the duodenal tube. It was clean golden yellow. Nevertheless, I thought that we must make a diagnosis of duodenal ulcer and gall bladder disease in addition. The patient was operated on, and a periduodenal abscess found. The gall bladder was not diseased. The liver was all right. The abscess was opened, and pus taken out. It was around the duodenum, which was thickened, leading to the perforation in the form of an abscess. The man was too weak to have anything else done, and Dr. Willy Meyer, who was the surgeon, decided that we could not do anything but drain the abscess. The man could not eat anything. He had severe pain and was vomiting, and the question was how to keep him alive. We both decided to introduce the duodenal tube two days after operation, in order to give him nutrition and let the ulcer heal up under rest. The man slowly got well. He had fever all the time for two or three weeks, due to the abscess. He is now well, with no symptoms, and has gained thirty pounds. That shows the importance of medical treatment in cases in which you see the lesion surgically. Very often patients are operated on for these craters that cannot be touched, and they get well afterwards, and the means that cure them are not the operation, but the possibility of giving them the desired attention.

Now, gentlemen, in conjunction with these remarks I will show you a procedure which may be employed when the duodenal tube is not able to pass the pylorus, on account of a stenosis of this outlet. I had such a patient not long ago with dilatation of the stomach, and ulcer and obstruction of the pylorus. The tube would not go through, and he was too weak for surgical intervention. I want to show you some means of arranging it so that the tube will pass in cases in which there is some stenosis, but not a complete stenosis. I have sometimes applied the same principle also in cases of dilatation of the oesophagus. It is to attach a bead by means of a silk thread about ten inches long to the end of the tube. The bead is surrounded by gauze. I found, years ago, that if we give a bead with gauze around it, the bead will pass more quickly into the intestinal tract, than the bead would alone. That is why I surround it with gauze. The bead is attached to the tube by means of the thread, and the patient swallows it. If the bead goes through the pylorus it sometimes pulls the capsule end of the duodenal tube through the pylorus. For two days the tube did not go in, even with using atropin, etc. Therefore, I made another attempt, using the bead. In twenty-four hours the bead went through, and we nourished the patient for two weeks with the tube.

He improved, and is now so that he eats liberally and has gained twenty pounds in two weeks. It is a very simple thing. I applied the same principle sometime ago for impermeable cardio spasmodic dilatation of the oesophagus. The pyloric dilator would not go in, but the bead did, and pulled the dilator through.

Dr. F. B. Baetjer, Baltimore, Md.: Mr. President and Members of the Society: I was particularly interested in Dr. Hamburger's paper, because in some work that Dr. Friedenwald and myself have been doing, we found the Roentgen examination of healing ulcers very important. In the paper we presented to the Association of American Physicians, in 1913, we called attention to that fact. We have had four or five hundred ulcer cases. Two hundred have been operated on. A number have been X-rayed, and we have obtained data that is trustworthy. We have checked up on the cases by means of the X-ray. We lay emphasis on the question of peristalsis. We always give the same sort of meal, and have the patient either fasting or on a full stomach. We always examine each patient under the same conditions. If you examine on a fasting and on a full stomach, you get different results. The X-ray will show conditions of increased peristalsis, though the defect may or may not be present. Some patients we put on a full meal, and found within a short time that the symptoms returned. In those cases, however, in which the clinical symptoms disappeared completely, and the X-ray symptoms disappeared, we found that when we put them back on ordinary diet, that they remained well over a given period of time. Sometimes patients come back at the end of one or two years with a return of symptoms. The defect, of course, is a very important thing, but we do not always get it in the marked degree that Dr. Hamburger has shown. I think he was dealing largely with chronic, long-standing, callous ulcers. In acute ulcers, we do not see the defect to the same degree, but get a much more marked peristaltic movement.

Dr. Mills raised the point of gastric and duodenal ulcers and the question of deformity. Our experience has been that in superficial ulcers the deformity is more general in the gastric than in the duodenal, that if we depended on the deformity in the duodenal, and not on peristalsis, we should be misled regarding cure. A certain amount of deformity may persist even with a clinical cure. I do not believe that the diagnosis of ulcer, its presence or absence, can always be obtained absolutely by surgical operation unless the stomach or duodenum is opened. We have had a number of cases, that on palpation revealed nothing, but that on exploration showed an ulcer to be present. Now we try in all cases of ulcer, to have a note made at operation as to whether the ulcer can be determined by palpation, and then as to the exact opening. We had a case that was of the acute type. The patient was passing a tremendous amount of blood. At operation, and palpation, the surgeon said, "Here is a small, indurated ulcer, which you can palpate." The duodenum was opened, and there was the ulcer. The surgeon had palpated one ulcer, and close to it was a mucous ulcer with a central point of bleeding. It was this ulcer that gave rise to the symptoms, and not the indurated ulcer near it. The indurated ulcer had given the deformity of the duodenum, but the peristaltic movements were out of proportion to the induration.

I wish to emphasize the point of Dr. Mills, which is very timely, namely, to see whether the patients get well or not by means of the radiographic examination. The plate is much more sensitive than the eye, consequently finer changes are recorded upon the plate than we can see in the fluoroscopic examination.

Dr. A. L. Benedict, Buffalo, N. Y.: I think that Dr. Johnson has made a very good point in speaking of the multiplicity of ulcers. So many times we use the term ulcer as if it were a proper, instead of a common noun. Five or six, possibly ten or a dozen kinds of solutions of superficial continuity may occur in the stomach. When we limit ourselves to peptic ulcer, Ewald's studies show about fifty per cent. of normal HCl acidity, twenty-five per cent. excessive, and twenty-five per cent. below normal. The process cannot be the same in all these instances. We have not got away from the idea that ulcer of the stomach is something eaten out by acid. It occurs to me that Dr. Hamburger's beautiful slides confirm the varied nature of gastric ulcers. Personally, I should consider myself very lucky to see so marked a projection at the site of an ulcer. I should consider it a rare case which we do not often strike practically. I should like to know whether the series of Dr. Hamburger consisted of selected cases, or whether the ulcer always shows so plainly by injection of the bismuth mixture.

It is interesting to recall that Dr. Hemmeter, in this country, was probably the first to call attention to the possibility of diagnosing gastric ulcer by the X-ray, although, of course, in a rather different way, by the lingering of the bismuth on the site of the ulcer.

You may remember that Dr. Hamburger showed a beautiful illustration of an ulcer and hour-glass constriction, not complete, which he afterwards explained was not accompanied with ulcer or hour-glass? I want to call attention to the fact that these beautifully typical hour-glass constrictions are the ones that are apt to be due to peristaltic waves. I had a patient with hyperchlorhydria and a perfectly normal stomach area, as determined by auscultatory percussion. The case was reported to me as one of hour-glass contraction. I looked at the plate, and found a beautiful hour-glass. The man who had done the radiography told me that he had another plate confirming that one. He showed me the other plate and, sure enough, there was another beautiful hour-glass. But what struck me as peculiar was that when the two plates were examined together, the hour-glass constriction shown was found to be in a different place in the two pictures. We then made a fluoroscopic examination, which showed that the condition was entirely functional, and not due to a lesion. So these very typical hour-glass contractions shown in the plates are the ones in which we should suspect a purely functional condition, due to the traveling of the peristaltic wave. Therefore, in ascertaining the gross contour of the stomach, I now depend on auscultatory percussion, rather than upon radiography.

Dr. Johnson, closing: First in answer to Dr. Lichty's question why I say so glibly that the thread may not be positive when the ulcer is in the splenic end of the stomach. I know this to be true because I have found it to be negative when the X-ray showed that ulcer existed and the finding was confirmed at operation.

In operations where the thread has been positive ulcer has been found in every case.

There is no single means by which comprehensive diagnosis of ulcer or of any other gastro-intestinal disease may be made. In my own work, after careful taking of the history, I employ physical, laboratory, fluoroscopic, roentgenoscopic, thread and every other available means, not only to detect the presence and character of the disease, but to determine as far as possible its primary or secondary relation to coexisting diseases, if there be any, and to all the conditions which may be associated with it. I do not believe that a thorough diagnosis can be made otherwise, or that treatment, internal or surgical, can be entirely and permanently successful.

A word or two in support of Dr. Hamburger's contention as to value of repeated examinations with the X-ray. I think that it should as he has said, be used, not only for confirmation of data already in hand, but for watching the progress of the disease and the effect of the treatment. I have very recently had a case, in which, at the first examination, a marked niche was found similar to the one he has demonstrated. After treatment of two or three weeks another exposure showed scarcely any defect, and the condition of the patient had correspondingly improved. By this means we are better able to direct our efforts and measure our results.

Dr. Hamburger, closing: I included in my complete paper the answers to at least some of the questions asked. I regret that I did not have time to read it in full.

In answer to the question regarding the depth of the ulcer and the danger of waiting for surgery, I would say that I think that this is a very proper question to ask. From our experience we think that the danger of perforation in deep ulcers is not so great as one would suppose. In ulcers of long standing, as most of these were, there is so much callous tissue thrown out that there was no *clinical* evidence at least of danger of perforation. The ulcers that were removed and examined could be held up to the light and one could see they did not penetrate to any great depth, and that there was considerable thickening of the mucosa and muscularis.

It was quite the exception in these cases to have hour-glass spastic contraction in association with chronic ulcers.

In regard to the question of malignancy, I have discussed this in somewhat more detail in the complete paper. This method of examination may throw further light on the frequency of metamorphosis of ulcer into cancer. I am inclined to feel that this does not occur so often as some believe, but I think that by examining ulcer patients in this way, from time to time, we shall gain much information on this point.

As to duodenal deformity, I should say, in general, that the duodenal ulcers that show the most deformity of the cap, exhibit, during the healing process, a gradual enlargement of the deformed area. I do not know what this is due to, but perhaps to the fact that part of the deformity is caused by edema and inflammatory exudate, which, during the healing, is absorbed. Several acute bleeding duodenal ulcers with no deformity of the cap, in the first examination after the hemorrhage, showed within twelve months a marked deformity. This is not necessarily an indication for surgery, but simply evidence of scar formation. If it does not interfere with duodenal function, leave it alone. If it does, surgery must be resorted to.

I should also like to speak of radioscopy. Plates are absolutely necessary for diagnosis. Many of our cases were overlooked by fluoroscopy, but the Nische was found by the plate, and later, by the help of the plate, we found the same thing with the fluoroscope. The persistence of the Nische, which Dr. Kaufmann mentioned, is valuable, likewise the fact that the recurrence of symptoms in chronic ulcer patients does not always mean an exacerbation of the old ulcer, but may mean a fresh ulcer. One of our patients had a recurrence of symptoms six months after treatment. The Nische did not recur. Until we found an abundance of blood and mucus in the stools and tenderness in the sigmoid, we did not discover that the symptoms were due to colitis, which, under proper treatment, disappeared.

The point that Dr. Einhorn brings up is valuable. I should like to ask what the obstruction was actually due to, in the case of pyloric obstruction with lesser curvature ulcer. If it were due to pylorospasm incident to the active ulcer, treatment of the ulcer would relieve the pylorospasm. If, however, it was due to organic stenosis, a gastroenterostomy would relieve this condition, and the ulcer would be only a secondary matter.

Dr. Baetjer's point is well taken. I agree with him thoroughly. Some cases we did check up on the X-ray evidence, by means of other tests. I think the plates are tremendously valuable, particularly in cases in which positive fluoroscopic findings are absent. You must then fall back on other things. In all the cases we checked up on the X-ray evidence by means of gastric secretion, blood in the stools, etc., etc.

Regarding Dr. Benedict's question, I would say that these were all selected cases. Indeed, they were very finely selected. We got them anywhere we could. They came from three different hospitals, and were unusual cases. They are probably quite rare, although I do think, gentlemen, that if you look for indurated ulcer without a Nische, you will discover that such ulcers are more frequent. They are not, however, frequent in any one person's individual private practice.

DR. MAX EINHORN, New York City: The thread test is based on the principle that when the string is tight, i. e., after having the pylorus, it can become impregnated with the secretions of the ulcerated area. When the thread is merely in the stomach, no imprint will occur. I did not think it necessary to refer to this, but I thank Dr. Hemmeter.

GASTRIC FUNCTION FOLLOWING GASTRO-ENTEROSTOMY. AN ANALYSIS OF 273 CASES.

By **Frank Smithies**, M. D.,

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To April 1, 1917, there had come under my observation 8,826 individuals affected with digestive disorders. Of this number, there were 273 patients (0.31%) upon whom gastro-enterostomy had been performed for the relief of dyspepsia. These patients returned for re-examination of their own accord or upon request, in order that their digestive functions might be ascertained, or they came because digestive upsets were troublesome. They represented 11.6% of 2360 individuals upon whom operations had been done for diseases of the stomach or the duodenum. These diseases were gastric ulcer 571 cases, duodenal ulcer 1469 cases and gastric cancer 320 cases. It is not to be inferred that the remaining 88.4% of patients in this group were well; no attempt has been made to trace them, inasmuch as this report is concerned with observations upon the functions of the gastro-enterostomized stomach, without particular effort being directed toward proving whether or no in the whole number of patients operated upon, the surgical procedure was the best type of therapy.

General Observations: Of the 273 patients composing this series, there were 170 males and 103 females.

The *average age* at which gastroenterostomy had been performed was for males 44.3 years and for females 41.5 years. When the patients returned for post-operative examination, the average age was for males 46.1 years and for females 44.3 years. It is thus evident that of the 273 cases, the males returned on an average of 1.8 years following operation and the females on an average of 2.8 years. The shortest interval intervening between operation and re-examination was, for males, 3 weeks and for females, 11 weeks. The longest interval between operation and re-examination was for males, 9 years and for females, 7 years.

The above facts exclude 58 cases that were studied post-operatively before leaving the hospital, at an interval of from 3 to 6 weeks following laparotomy.

Primary Operative Procedures.—As table II demonstrates, posterior gastroenterostomy, without or with pyloric closure or local or extensive gastric resection and combined with removal of the appendix or removal or drainage of the gall bladder, was the operation of choice in 95.4% of cases. In 4.24% anterior gastro-enterostomy was similarly employed. In one patient (0.36%), through faulty anatomical orientation, at another clinic, a gastro-ileostomy had been done under the impression that a gastro-enterostomy was being performed. This patient survived nearly one year.

Patients Subjective Anomalies of Digestion at Re-examination.—It should be again emphasized that 58 patients were examined within six weeks following operation. It is not to be expected that perfect digestive function had become established in so brief a time. However, reference to Table III shows that of 57 gastroenterostomized patients (20.9% of the series) clinically, complaint free, there were 18 such whose operations had been very recent. Moreover, of 78 patients (28.9%) who were subjectively in good health, but who experienced mild digestive upsets, (usually stated as being "dietetic"), there were 26 or one third of the group, whose laparotomies had taken place within 6 weeks. Of the entire series (273 cases), 104 patients (38.09%) were definite dyspeptic, although they stated that operation had been followed by improvement. This class includes 9 patients (9.6%) recently operated upon. There were 20 patients (6.9%) in whom gastro-enterostomy has been followed by no improvement whatever in digestion, and upon three of this class the operation had been performed less than six weeks previous to examination. Fourteen patients (5.1%) appeared to have experienced an aggravation of their dyspepsias post-operatively. Of the two recently operated patients in this group, upon one had been performed simple posterior gastrojejunostomy without pyloric closure on account of non-stenosing pyloric ulcer and upon the other posterior gastrojejunostomy had been combined with resection of the pyloric fifth of the stomach for *ulcus carcinomatosum*.

It is evident from the study of Table III that of the 273 gastro-enterostomized patients re-examined, but 20.9% were complaint free; 49.8% were clinically comfortable, and in 87.89% subjective benefit had accrued from the operation. If the 58 patients recently operated upon are deducted on the ground that it is as yet too early to establish their status, there still remain 196 patients (68.49%) who have been subjectively improved by gastro-enterostomy and associated surgical procedures.

Post-Operative Symptoms in Patients Not Dyspepsia-Free.—There were 216 such patients. The type of their post-operative digestive upsets and their clinical symptoms are analyzed in detail in Table IV. It has seemed advisable to group the symptomatology according to the gastric or the duodenal ailment for which operation was performed. The summary makes apparent that where the surgery had been done for relief of cancer and of post-pyloric duodenal ulcer, abdominal distress, anorexia, nausea, vomiting, gas, "water-brash," and constipation, were more frequent than where the operative indication had been gastric or pyloric ulcer. Eructations of food and diarrhoea were rather more frequently observed in the gastric ulcer cases. It should be emphasized that very few gastric cancer patients returned for re-examination following operation. They were all in a bad way; what became of the original group (320 cases) of which they formed a part, it is not possible to state. Doubtless, fully half of the original group is dead. Weight loss had been experienced by nearly one-sixth of the gastric or pyloric ulcer class, by more than one-fourth of the duodenal group and by all of the cancer patients.

The incidence of gross hemorrhage (hematemesis or melena) following the operative procedures warrants consideration. It would apparently indicate delay in healing, recrudescence of the original ailment, malignant change or the occurrence of new pathologic processes (doubtless, most commonly ulcer). In more than 6% of the gastric or pyloric ulcer cases, more than 17% of the post-pyloric duodenal ulcers and approximately 16½% of the gastric cancers, gross hemorrhage had occurred post-operatively. In 6 patients, the hemorrhages were exhibited within four weeks following the laparotomy. It is customary to ascribe such bleeding to faulty operative technique or to accidents. It is certainly not possible always to explain the event in this way. The occurrence of gastrorrhagia following surgical procedures upon the appendix, gall-bladder or pelvic organs, might indicate causes for such hemorrhage, apart from those purely mechanical.

Signs of Digestive Mal-Function: (Table V) Less than one-third of the patients exhibited gross physical evidences of digestive anomaly. Of 68 patients whose blood was examined in but 30.8% was the *hemoglobin* below 80%. There was nothing especially noteworthy in the blood counts. Rather more than 10½ of the patients were definitely *cachetic*. This is not remarkable, when it is recalled that there were 12 cases of hopeless gastric cancer in the series. *Jaundice* was distinctly present in 11 cases (4.03%). In 5 patients there was malignant disease involving the pancreas, gall tract or liver. In the remaining 6 cases, post-operative adhesions, perforated ulcer involving the pancreas, gall-stones and biliary cirrhosis explained the jaundice. *Visible abdominal peristalsis* was observed in 19 patients (6.9%). It seemingly resulted from gastric hypermotility associated with stenosis of either the pylorus or gastro-enterostomy stoma or both or occurred in the jejunum as a consequence of adhesions, or imperfect surgical manoeuvres. In the patient upon whom gastro-ileostomy had been performed, distension and billowing of the abdomen were striking. Fecal vomiting was a daily event. *Abdominal tenderness* was recorded in 82 patients (30.0%). It was constant in those patients examined soon after operation. In the patients whose gastro-enterostomies had been performed at intervals of from 1 to 5 years, acute abdominal tenderness was noted rarely. Tenderness appeared to be commonly due to gastric hyperaesthesia (diffuse gastritis?); recurrence of ulcer, gastric cancer, gaseous distention of stomach or jejunal loop and in two instances to jejunal ulcer. *Abdominal ridge or mass* was observed in 44 cases (16.1%). Of these there were 11 patients affected with gastric cancer. In the remaining cases the tumor was apparently produced by persistent abdominal wall scar, adhesions of the viscera to the parietal peritoneum, local peritonitis about the operative field, exhuberant scarring at the side of the gastro-enterostomy or the point of gastric resection, persistent ulcer scar, enlarged pancreas, inflamed lymph nodes and rolled up omentum. In one case, a Murphy button had become partly detached and was fixed just beyond the gastro-jejunal stoma.

Observations Upon the Size or Capacity of the Stomach were made by the air inflation method in 179 cases. It is recognized that such method is only relatively accurate. However, the air inflation is as reliable as are other accepted procedures. An air bulb of 1½ ounces (water capacity) was used. The average capacity of the normal male stomach was considered as 33 ounces, and that of the normal female stomach as 27 ounces, as demonstrated by me[1] from the examination of 7,041 individuals in 1914. It will be observed (Table V) that in 47.4% of the gastroenterostomized patients the gastric capacity was below normal, in 20.6% larger than normal, and in 31.9% not appreciably changed. These facts are of value because it has been generally presumed that gastro-enterostomy brought about definite reduction in stomach size as a consequence of its "drainage" effect. Dietetic regimes have not often been based upon this supposition. It is, therefore, interesting to note that despite pyloric resection being associated with gastro-enterostomy in 10.59% of our cases, less than one-half of the stomachs appear to be below normal capacity. The factor of rapid escape of air through the gastro-enterostomy stomach or through this and the pylorus, contributing to an apparent increased capacity of the stomach does not appear to warrant serious consideration. It is demonstrated by the fluoroscope and by test meals that a sphincter-like arrangement commonly exists at the gastro-enterostomy stomach as well as at the pylorus, and that air inflation tends rather to close the gastro-enterostomy stoma than to force it open; that is, provided the stomach is not greatly over-inflated. Air inflation rarely results in dilatation of the jejunal loop.

Test Meal Observations—(Table VI). The emptying rate of the gastro-enterostomized stomachs was estimated physiologically, *i. e.,* the patient was permitted to eat a full-sized meal of foods that he liked, with the proviso that meat, lettuce (or celery or raisins) were included. The stomach was emptied after 8 hours by a large calibre stomach tube and afterwards lavaged with 2 liters of warm water in order to avoid missing retention food bits. The observations of Levy and Kantor[2] upon 1600 patients, and of ourself[1] upon 7041 patients have abundantly demonstrated that food must persistently remain for longer than 7 hours in order to establish proof that definite stagnation exists. Few stomachs are entirely food free within 7 hours after a hearty meal of mixed food.

Of 262 patients in this series whose stomach emptying rate was examined persistent *food stagnation,* as above defined, was demonstrated in 43 or 16.4%. It was present in 10 of the 12 cancer cases in the series. *Gastric hypersecretion, i. e.,* the recovery of more than 150 c.c. of contents following an Ewald meal—was proven in 29 cases, or 11.06%.

Of the entire series the average quantity removed from the stomach was for the stagnation cases 347 c.c. and for the non-stagnation cases 126 c.c.

Chymification was "good" in 56.6%—"fair" in 33.3% and "poor" in but 10%. This observation is not without interest, in view of the criticism which is often made that gastro-enterostomy markedly alters gastric peristaltic activity, as demonstrated by the Roentgen ray. It is suggested that real food acts differently upon gastric mechanism than does an inert heavy mass of bismuth or barium.

Bile staining of the gastric extracts was definite in 57.7% of meals examined. The colors ranged from a light golden yellow to grass green or olive. It is interesting to observe that even in high acid meals, bile coloring was frequently intense—in fact, the macroscopic presence of bile coloring was no index of the degree of gastric acidity, either free HCl or total.

Nearly one-third (30.5%) of the gastric extracts were uncolored except as the shade varied with the test meal given. In 11.8% of meals, discoloration resulted from food remains, cell detritus, bacterial or ferment action and possible altered bile or blood.

Macroscopic blood was present in 10.7% of the stomach contents. It was apparently of traumatic origin.

Observations upon Gastric Acidity.—Inasmuch as numerous workers, particularly Bolton[3] and Paterson[4] have emphasized the importance of hydrochloric acid in the causation and the healing of peptic ulcer, comparison has been made between the test meal findings respecting acidity before and after gastroenterostomy. (Table VII.)

It will be observed that there occurred no post-operative decrease in free HCl in 14% of cases, and that in 5% there was a definite increase. There was a *demonstrable reduction in free HCl* in 81% of all cases examined. This reduction averaged 20.5%. The table also demonstrates that the reduction in free

HCl was greatest soon after operative procedures and that there was a steady diminution in the degree of acid reduction in direct proportion to the time interval following operation. The greatest average reduction (37) was recorded in those cases exhibiting "vicious circle."

Respecting *Total Acidity*, it is evident from Table VII that there occurred *no post-operative reduction* in 17% and that there existed a post-operation increase in 3%. In 80% of the cases there was, however, definite *post-operative reduction in Total Acidity*. It averaged 21.02. The greatest degree of reduction occurred soon after laparotomy. In general this degree became less pronounced as longer time intervened between operation and re-examination. Cases exhibiting "vicious circle" showed a Total Acidity reduction averaging 28.

The above facts clearly demonstrate persistent reduction in both free HCl and total acidity following gastrojejunostomy. If excessive concentration of free HCl is an important factor in the production of and the prevention of healing of peptic ulcer, then it might be assumed that gastro-jejunostomy, properly used, holds valuable therapeutic possibilities. What effect upon the reduction of acidity and towards the healing of peptic ulcer bile (noted macroscopically in 57.7% of this series and chemically proved to be present by Goodel's test in 42% of 182 meals analyzed) exerts in open to question. Paterson, in a rather indefinite research, stoutly maintains that bile (mixed with pancreatic juice) is "present almost invariably, in the stomach after gastrojejunostomy," and that such is present in "less than 10%." He states that the reduction in total acidity averages 30%, and that this is "partly due to neutralization of free hydrochloric acid by bile and pancreatic juice and partly to earlier stimulation of the pancreatic secretion and compensatory (!) earlier lessening of the gastric secretion." Paterson also states that after gastrojejunostomy there "is an almost constant increase in the mineral chlorides of the gastric contents" and as a rule "a diminution of the total chlorides." The "increase in the mineral chlorides disappears" after undoing a gastro-jejunostomy. He claims that the average increase in the mineral chlorides is 0.077%, and that such increase is evidently due to bile and pancreatic juice gaining access to the stomach through the amastomotic opening. It would seem that according to Paterson, this increase in mineral chlorides is an important factor in reducing gastric acidity, and thus aiding ulcer healing. However, Panton and Tidy[5] maintain as the result of much painstaking research upon the value of various methods of gastric analysis, that there is no really reliable technique by which mineral salts (chlorides, phosphates) can be accurately estimated in gastric contents. They assert that in low acidity cases the amount of phosphates present is probably independent of the clinical condition, and that phosphates introduce a "fallacy" common to all methods investigated." Further that when free HCl is low or absent, the "active hydrochloric acid" is overestimated because the fixed chlorides interact with phosphorous compounds, with the consequence that the phosphates are increased. Panton and Tidy also suggest that phosphates in excess are secreted into the gastric cavity, when the stomach mucosa fails to elaborate free HCl from the mixture of phosphates and chlorides brought to it from the blood. It would thus appear that Paterson's opinion respecting the source of increased mineral chlorides in gastro-enterostomized stomachs is open to question, and that even the increase which he maintains exists (0.077%) is well within the possibility of chemical error. Hamburger[6] has recently confirmed the work of Schutz[7] and Levites[8] with reference to the inhibiting action of various alkaline salts on peptic digestion. He claims that such alkalies definitely inhibit peptic digestion and, in a later communication with Halpern,[9] suggests that inasmuch as phosphates, chlorides and carbonates inactivate pepsin, a therapeutic advantage in the cure of peptic ulcer can be secured by their use. Inasmuch as it seems evident that phosphates (and perhaps chlorides) are increased in gastro-enterostomized stomachs as free HCl is decreased, it would appear that the experiments of Hamburger and Halpern might explain some of the beneficial effects of gastro-enterostomy toward ulcer cure. It is still unsettled whether bile and pancreatic juice contain "antipepsin" when regurgitated into the stomach. Bile was present in 9.5% of the gastric contents from 140 non-stenosing ulcer cases that I analyzed—and yet two-thirds of the ulcers lay in the pyloric fourth of the stomach where they could secure the maximum benefit of bile regurgitation should it contain "antipepsin."

Further Chemical Observations Upon Test Meals—(Table VIII).—As I have already mentioned *bile* was proved to be present in 42% of the gastric extracts from 182 gastro-enterostomied stomachs (Goodel's Test).[10]

Altered blood was demonstrated in 42.5%; *lactic* acid in 3.4% (mainly the cancer cases) and *volatile fatty acids* in 2%. (Also the cancer cases.)

Wolff-Junghans'[11] test for soluble albumin was negative in nearly three-fourths of the cases.

The *Formol Index*[12] demonstrated a slight increase over the creptic power common to peptic ulcer, but no increase over that observed in cancer. It averaged 14.2% in 81 cases analyzed. Such increase might be taken to indicate the presence of pancreatic juice, although the rather high percentage (45.2) of extracts exhibiting positive altered blood test, may explain the increase.

Microscopic Examination of Gastric Extracts exhibited nothing characteristic. In the benign stagnation extracts yeasts and sarcinae were often abundant. In the malignant retention extracts organisms of the Oppler-Boas group were plentiful. Regurgitated bile and pancreatic juice appears to have no effect upon the gastric flora. In 5 cases where stagnation existed in the jejunal loop, the presence of a peculiar, short fat, deeply staining, acid-fast bacillus appeared constant. When it had been observed several times, its presence in large numbers led to the diagnostic suggestion that jejunal stenosis with dilatation might be present, which suggestion was substantiated at laparotomy.

Stool Analyses.—Altered blood was demonstrated by the benzidin test in 31 of 107 cases studied. Of these 72 patients were upon test diet.

To 28 patients Schmidt's test diet was given. In 17 cases (60.7%) there was evidence of deficient proteolytic digestion.

Bile Pigment (Schmidt bi-chloride method) was present in all but 8 of 151 stools examined, either as hydrobilirubin or biliverdin. In 7 instances where bile pigment was absent there was deep jaundice. In the remaining case there existed intestinal obstruction and pronounced cachexia (instance of gastroileostomy).

Roentgen Examination.—Fluoroscopic study was made of 39 cases within 7 weeks of gastro-enterostomy and of 26 cases operated upon from 6 months to 9 years previously. The results were so greatly at variance with the patient's physiologic digestive function or his clinical condition that we are still in doubt as to the clinical worth of the procedure. Apart from the screen and plate examinations demonstrating the patency or closure of the pylorus or gastro-enterostomy stoma, contractures of the stomach, incorrect surgical procedures, new ulcer or recurrences of ulcer or cancer, or anomalous pocketing or regurgitation of the opaque meal, little reliable information respecting gastric function was obtained. Many of these facts were clinically evident from simple routine examinations and from well taken histories.

In patients who have been operated upon at other clinics and in whom the surgical manoeuvres are in doubt, fluoroscopic examinations furnish a valuable method for rapidly establishing the mechanical status of the stomach and the jujunum. However, in all cases, where stenoses, contractures, pocketings or retention of the opaque meal appear to exist, frequent examinations, particularly after full doses of atropin or belladonna, should be made before opinion is given. Many abnormalities seen at the first seance disappear upon repeated examination, especially upon the examination after antispasmodic medicines have been administered.

REFERENCES.

[1] Smithies, F.: *American Journal of the Medical Sciences*, 1915, October, p. 1,041.

[2] Levy, T. H., and Kantor, J. L.: *Arch. Int. Med.*, 1916, April, p. 476.

[3] Bolton, Ch.: *Journal of Pathology and Bacteriology* (British), 1915, Vol. XX, p. 133.

[4] Paterson, H. T.: Proceedings XVIIth International Congress of Medicine, Section of Surgery, London, 1914.

[5] Panton, P. N., and Tidy, H. L.: *Quarterly Journal of Medicine*, 1911, July, p. 449.

[6] Hamburger, W.: *Arch. of Int. Med.*, 1915, Sept., p. 356.

[7] Schütz: *Beitr. z. chem. Physiol. u. Path.*, 1904, V, p. 406.

[8] Levites: *Ztschr. f. physiol. Chem.*, XLVIII, p. 187.

[9] Hamburger, W., and Halpern, B.: *Arch. of Int. Med.*, 1916, August, p. 228.

[10] Goodel, J.: *Boston Med. and Surg. Journ.*, 1911, p. 780.

[11] Smithies, F.: *Am. Jour. Med. Sc.*, 1914, February, p. 360.

[12] Sorensen-Schiff: *Zeitschr. f. physiol. Chem.*, 1906, p. 712.

TABLE I.

SUMMARY OF MATERIAL STUDIED.

Total cases examined		8826
Number of cases operated in that time for	Gastr. ulc.	571
	Duod. ulc.	1469
	Cancer	320
Number cases examined after gastro-enterostomy		273 (.31%)

AILMENT CALLING FOR PRIMARY OPERATION.

226 following operation for gastric or pyloric ulcer.
12 following operation for gastric cancer.
35 following operation for duod. ulc. not involving pylor.

Sex	Males,	170
	Females,	103
Average age at operation	Males,	44.3 years
	Females,	41.5 years
Average age when re-examined	Males,	46.1 years
	Females,	44.3 years
Average length of time following G. E.	Males,	1.8 years
	Females,	2.8 years
Longest interval	Males,	9.0 years
	Females,	7.0 years
Shortest interval	Males,	3.0 weeks
	Females,	11.0 weeks

58 cases studied before leaving hospital, i. e., 3 to 6 weeks following operative procedure.

TABLE II.

OPERATIVE PROCEDURES.

Type of Operation.	No. of Cases.	Per Cent.
Post. gastrojejunostomy	91	33.3
Anterior gastrojejunostomy	5	1.8
*Post. Gast.-ent. with operative pyloric closure	50	18.3
Post. Gast.-ent. with resection of ulcer or part of stomach	26	9.5
Ant. Gast.-ent. with resection of ulcer or part of stomach	3	1.09
Post. Gast.-ent. with appendicectomy	38	13.9
Post. Gast.-ent. with cholecystectomy	14	5.1
Post. Gast.-ent. with cholecystostomy	26	9.5
Post. Gast.-ent. append. cholecystect	8	2.9
Post. Gast.-ent. append. cholecystost	8	2.9
Ant. Gast.-ent. appendicectomy	2	0.73
Ant. Gast.-ent. cholecystostomy	1	0.36
Gastroileostomy appendicectomy	1	0.36
Totals	273	99.74

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*17 cases of pyloric closure by suture or fascial bands.

TABLE III.

CLINICAL CONDITION OF PATIENTS.

Status	No. of Patients.	Per Cent.
Clinically complaint-free (includes 18 patients recently operated)	57	20.9
Well, but with minor digestive upsets (includes 26 patients recently operated)	78	28.9
Improved, but still dyspeptic (includes 9 patients recently operated)	104	38.09
Not improved (includes 3 patients recently operated)	20	6.9
Dyspepsia aggravated following operation (includes 2 patients recently operated)	14	5.1
Total	273	99.89

SUMMARY.

Clinically comfortable	145	49.8
Improved	104	38.09
Not improved	34	12.0
Or		
Subjectively benefited	249	87.89
Deducting 58 patients (19.4 of total) recently operated leaves		
Subjectively benefited	196	68.49

TABLE IV.

POST-OPERATIVE SYMPTOMS IN THE PATIENTS NOT DYSPEPSIA FREE.

Complaint.	Gast. or Pyl. Ulc. (226)		Duoden. Ulc. (35)		Gastric Cancer (12)	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
Pain or distress	132	61.5	28	80	12	100.0
Anorexia	27	12.8	7	20	9	75.0
Nausea—constant or irregular	41	18.9	14	40	8	66.6
Vomiting	43	19.9	7	20	10	83.3
Stagnation type	20	9.2	4	11.4	6	50.0
Gas, bloat, etc.	67	31.0	14	40	11	91.7
"Water brash"	34	15.7	8	22.8	5	41.6
Eructations	95	43.9	12	34.2	11	91.7
Constipation	44	20.3	8	22.8	9	75.0
Diarrhea	19	8.9	3	8.5	2	16.6
Weight loss	35	16.2	10	28.5	12	100.0
Weakness	39	18.0	8	22.8	12	100.0
Nervous	18	8.2	4	11.4	5	41.6
Gross hemorrhage	13	6.01	6	17.1	2	16.6

TABLE V.

OBJECTIVE EVIDENCES OF DIGESTIVE MAL-FUNCTION.

Sign	No. of Cases.	Percentage
Anemia (Hg. 80% or lower, of 68 cases examined)	21	30.8
Cachexia	28	10.3
Jaundice	11	4.03
Visible Abdominal Peristalsis	19	6.9
Abdominal Tenderness	82	30.0
Abdominal Ridge or Mass	44	16.1

OBSERVATIONS UPON STOMACH SIZE.

Inflation Method.

Stomach smaller	85, or 47.4%	Of 179 cases tested.
Stomach larger	37, or 20.6%	
No change	57, or 31.9%	

TABLE VI.

TEST-MEAL OBSERVATIONS.

Gastric stagnation (longer than 8 hours after full meal)		43 cases, or 16.4 per cent.
Gastric hypersecretion (more than 150 c.c. recovered, exclusive of Group 1)		29 cases, or 11.06 per cent.
Average quantity removed	Stagnation cases	347 c.c.
	Non-stagnation cases	126 c.c.
Chymification	Good	56.6 per cent.
	Fair	33.3 per cent.
	Poor	10.0 per cent.
Bile stained gastric extracts		57.7 per cent.
Discolored gastric extracts		11.8 per cent.
Uncolored gastric extracts		30.5 per cent.
Macroscopic blood		10.7 per cent.

TABLE VII.

OBSERVATIONS UPON GASTRIC ACIDITY.

Free Hydrochloric.

No post-operative *reduction*	14 per cent.
Post-operative *increase*	5 per cent.
Reduction in cases examined *soon* after operation *averaged*	35.0
Reduction in cases examined *1-3 years* after operation *averaged*	22.0
Reduction in cases examined *3-5 years* after operation *averaged*	14.0
Reduction in cases examined *more than 5 years* after operation, *averaged*	11.0
General average of acid reduction	20.5
Reduction in cases exhibiting "*Vicious Circle*" *averaged*	37.0

Total Acidity.

No post-operative *reduction*	17 per cent.
Post-operative *increase*	3 per cent.
Reduction in cases examined *soon* after operation *averaged*	42.0
Reduction in cases examined *1-3 years* after operation *averaged*	13.5
Reduction in cases examined *3-5 years* after operation *averaged*	18.0
Reduction in cases examined *more than 5 years* after operation, *averaged*	10.6
General average of acid reduction	21.02
Reduction in cases exhibiting "*Vicious Circle*" averaged	28.0

TABLE VIII.

OTHER CHEMICAL TEST-MEAL OBSERVATIONS.

Bile (Goodel's test)		42.0%	(182 tested.)
Altered blood (Benzidin test)		45.2%	
Lactic acid (ether extract)		3.4%	
Volatile fatty acids (heat test)		2.0%	
Wolff-Junghan's test:	Positive	7.1%	Of 66 cases tested.
	Questionable	18.0%	
	Negative	74.9%	
Formol index (Sorensen-Schiff method) averaged		14.2	Of 81 cases tested.

DISCUSSIONS.

Dr. Julius Friedenwald, Baltimore, Md.: About three years ago, Dr. Finney and I reported our experiences with one hundred cases of pyloroplasty, and a year ago in one hundred cases of gastro-enterostomy. We compared the results after both operations and observed that the immediate effect of both procedures was quite good; but that the favorable result was not quite immediate, the stomach requiring two or three months before returning to normal function. A normal motility was not secured until two or three months after the operation. This was often delayed further by injudicious feeding. The end results which we obtained, that is as long as five years after operation, were much in favor of pyloroplasty over gastro-enterostomy. In the pyloroplasty cases, the percentage of satisfactory results was 88 while in gastro-enterostomy it was 77.

Dr. J. C. Hemmeter, Baltimore, Md.: It is very curious what a fascination this subject has for the clinician. We have had a number of symposiums on it; at least two, I think. This fascination comes from the fact that, on the whole, the pathology of the digestive tract is something that is of interest to both sides of the medical profession —the surgeons, as well as the clinicians.

TABULATION.

Among the pictures thrown on the screen by Dr. Smithies was one containing some figures, in connection with which I must speak of a fact that must have impressed you all, at a glance. You will find no German, French or American textbook which makes a claim of recognition of gastric ulcer in more than one-eighth per cent. of all cases. Dr. Smithies, however, was able to make the diagnosis in one-fourth per cent., or over. In other words, we are now able to make a diagnosis of more than one-fourth of the total material; whereas, fifteen years ago, no one claimed to be able to do so in more than one-tenth to one-eighth per cent. I think that this shows a very good progress, on which we should all congratulate ourselves; and it is largely due to the work done by members of the American Gastro-Enterological Association. At the date of the Twentieth Anniversary of this Association, it is well to call attention to the work of our members. The name of Turck was forgotten, a while ago; and another man was mentioned, who worked long after him.

I am requested to explain something that I thought Dr. Einhorn would explain. Dr. Lichty asked a question of Dr. Johnson, and Dr. Johnson agreed that you cannot get a positive string-test in what he calls the splenic end of the stomach. Why is it that a positive test could not be obtained in cases in which the ulcer was in that location? Fourteen years ago I made string-test experiments on animals with artificial gastric ulcers, and we passed the needle through the cheek, so that the animal would not chew the string. The string always takes the shortest route. It is pulled on, and cannot get into the splenic flexure. It is tight, and not slack. We need not, however, be discouraged on that account; because the pathologist tells us that the lesser curvature and the pylorus contain seventy per cent. of all ulcers.

DR. R. WALTER MILLS, St. Louis, Mo.: I wish to say just a word regarding the thread test, which everyone seems to be surprised to find efficacious. The stomach, when empty, is a contracted tube, and folds together in much the same fashion as does an umbrella. Through capillary attraction there may be a local accumulation of the secretions at the ulcer level; so that it is immaterial whether the thread actually crosses the ulcer or not. The only place that the secretions would not be in contact with the thread is in the fundus, which is occupied by bubbles of gas.

DR. SMITHIES (closing): As Dr. Hemmeter stated, we have had a good many symposiums on the subject of gastro-enterostomy; but these have been lessened in value, because they were usually a fight of the medical men against the surgical, as to whether it was a good procedure. I said in my paper that I made no claim as to whether such operation was a beneficial procedure or not. I thought that the society was ready for a simple statement of fact as to what happens when it is undertaken. That I endeavored to make in the brief summary of the paper that I presented. Probably someone with a more active imagination than mine will be able to deduce from these facts just what benefit the patients who returned had secured from the operation. We did not have an account from the great group of patients who had been operated on and had not returned. Some had no doubt died, or had drifted to other clinics, or had taken to Christian Science or crystal gazing.

We have a follow-up system. The patients have cards given them, which they mail back every six weeks to three months These cards have on them questions that the patients are to answer, and on these answers we base our evidence.

I wish to explain the plan on which we are basing diagnoses. We always record the histo-pathologic diagnosis. Of the cases, all were operated and proved to be either ulcer or cancer. The material that we handled was end-result material. There was no brilliancy in the diagnosis, unless it was exhibited by the surgeon or the pathologist. We thought that it would be interesting to report these cases, because they form the largest group of cases that to my knowledge has ever been analyzed.

REPORT OF A CASE OF HUGE DILATATION OF THE ESOPHAGUS.

By **Julius Friedenwald**, **Albertus Cotton**,

and

A. C. Harrison,

BALTIMORE.

Cases of diffuse dilatation of the esophagus are not uncommon; but the case which we are about to report had a number of unusual features making it of considerable interest.

B. F. C., age 40 years, a brakeman by occupation, was referred to us by Dr. Z. K. Wiley on December 6th, 1916, and admitted to the Mercy Hospital. The patient complained of inability to retain food, gradually increasing weakness, and eructations. His family history was negative; there is but little of interest in his personal history. He fell from a train in 1907 and fractured two ribs on the left side; he had been suffering for many years with headaches and constipation; his appetite has been good; the bowels are constipated; there has been neither hematemesis nor melena. After the ingestion of a meal the patient has had to make repeated swallowing efforts in order to prevent the food from being regurgitated. Soon after eating eructations are produced, and the food is regurgitated, a mouthful at a time, lasting from two to four hours, and sometimes as long as eight or ten hours. He finds that he can swallow solids as well as liquids, but more discomfort is produced by solid food; occasionally vomiting of food eaten the day before occurs. The patient suffers at times with epigastric pain, radiating to the back between the shoulder blades, which he ascribes to gas; he is usually, however, free from pain. Food taken can be reguritated at will. He feels full and bloated after eating, but this condition is relieved by eructations or by vomiting. Nausea is frequently present and the patient complains of a bad taste in the mouth.

The patient had been a brakemen for nineteen years, and has been drinking three or four cups of coffee a day, he has not been addicted to the use of tea, beer or whisky; he smokes three cigars a day.

Present Illness: The patient was perfectly well and had no digestive disturbance until his twenty-second year. At this time after having partaken of a meal consisting of ham and eggs, indigestion set in, accompanied by pain in the abdomen and vomiting. The patient dates the onset of his trouble to this attack. Since then he has been regurgitating food and has had indigestion more or less most of the time. His condition has gradually become aggravated although he has periods of complete relief from symptoms lasting a month or two at a time. He has gradually been growing weaker. When the indigestion is especially severe the skin becomes dry and cracked. The patient claims that he can eat with ease two-thirds of a loaf of bread, two eggs, two glasses of water and two cups of coffee; but in several hours regurgitation occurs. The appetite is good and is rarely satiated. The patient has lost about forty pounds in weight in the last six months.

On physical examination the patient presents a pallid complexion and is much emaciated; his cervical glands are enlarged. The lungs are apparently normal, vocal fremitus is, however, somewhat increased on the right side and the percussion note appears to be of a higher pitch than that over the left side. The apex beat of the heart is found in the epigastrium just beneath the ensiform cartilage; the heart is apparently displaced downward and to the right.

On examination of the abdomen no masses are observed, the veins are enlarged and plainly visible; the liver and spleen are not enlarged and there are no areas of tenderness. The external abdominal rings are enlarged and small hernias are present on both sides.

The urine presents neither sugar nor albumin. It contains a few pus cells, oxalate of lime crystals and a few hyaline casts. The blood examination reveals a total red count of 3,540,000; white count, 7300; hemoglobin, 65%. The differential count is as follows:

Polynuclears	70%
Small mononuclears	23%
Large mononuclears	6%
Eosinophiles	1%

The Wasserman reaction is negative.

The stool is soft, brown, alkaline in reaction, contains traces of occult blood and triple phosphate crystals. Neither parasites nor ova are present.

The contents of the esophagus was removed one hour after an Ewald test breakfast; 530 c.c. of contents were obtained, white in color of a total acidity of 25 and with an absence of free HCl. with mucus and lactic acid in abundance and also containing occult blood. Microscopically the contents contains starch granules, yeast cells and Oppler-Boas bacilli. Twelve hours after a retention meal 650 c.c. of contents were removed presenting the same characteristics as have been already described. The capacity of the esophageal dilatation was found to be 1700 c.c.

An X-ray examination revealed an enormous dilatation of the esophagus with marked retention. This retention was most marked for 24 hours, and to a slight degree even for 48 hours. The barium could be seen trickling through an exceedingly narrow orifice. The retention was so great that it was difficult to obtain any satisfactory plates of the stomach itself, for the small quantity of barium which would trickle into the stomach would pass out almost as quickly. Small quantities of barium were, however, observed in some plates far down in the abdomen pointing to a gastroptosis.

A diagnosis of diffuse dilatation of the esophagus was reached and attempts were made to pass through the strictured area. At first an Einhorn duodenal tube was introduced, but failed to pass the cardia; the patient was then allowed to swallow a thread, which gradually found its way through the cardia into the stomach; gradual dilatation being contemplated, when the string had passed sufficiently far to be used as a guide for the passage of perforated olive bougies. After frequent attempts, it was observed that the thread would not pass through the stomach notwithstanding the fact that it was left in situ at various periods for from 24 to 48 hours. This was due to a marked pylorospasm, as was subsequently observed at operation. The patient was exceedingly weak and so emaciated that further delay was no longer considered safe, and it was deemed best to open the abdomen and dilate the cardia from below.

On December 15th a gastrotomy was done under rectal ether anesthesia. The stomach was opened on its anterior surface near the cardia and the silk thread which had been previously swallowed, was delivered. With a little effort the index finger of the operator was forced through the cardia into the esophagus and bougies of various sizes including the largest were passed. In as much as there was but little difficulty in finally passing the largest bougies, and on account of the danger of infection due to the great amount of manipulation in the stomach, it was deemed advisable to close the stomach. For a few days the patient was able to swallow, and was free from attacks of vomiting, but was soon again unable to retain any food. It was, therefore, deemed advisable to perform a gastrostomy which was done on December 23rd under local anesthesia. The patient was fed at once with small quantities of fluids through the gastrostomy opening. He was exceedingly weak, however, and did not rally and died on December 26th.

Autopsy Findings: The thorax and abdomen were opened in the usual manner. The left thorax was normal; when the right lung was pushed aside the enormously dilated esophagus was revealed. The esophageal dilatation was fusiform in shape, the smaller portion of which extended as high up in the neck as the cricoid cartilage. The esophageal sac held as much as 1750 c.c. of water. The stomach was about normal in size, with a capacity of 750 c.c. The other abdominal organs were normal. This case presents a number of rather interesting features; the most important of which are first, the very extensive dilatation of the esophagus, and second, the inability of the silk thread to pass through the pylorus.

While cases of idiopathic dilatation of the esophagus due to cardiospasm are not infrequent, it is unusual to observe cases presenting dilatations so extensive as the one reported, the capacity of the esophagus being 1,750 c.c., over twice as great as that of the stomach. It is unusual, too, no matter how marked a stricture might be to observe that a silk thread will not seek its way through the stomach into the bowel, it being well known that a thread will find its way through any passage that water will. In our case this difficulty was demonstrated to be due to pylorospasm. It is quite probable that if the patient had been examined at a less advanced stage of his affection, it might have passed without difficulty. Inasmuch as the passage of the thread is the most important feature preliminary to proper treatment of this conditon, the usual treatment that is dilatation by the passage by perforated olive bougies over the thread drawn taut could not be practiced.

The question as to the etiology of idiopathic dilatation of the esophagus has been a matter of some discussion, especially regarding the question as to whether the cardiospasm precedes the dilatation or follows it. Both Strumpel and Mikulicz recognized the importance of cardiospasm in the development of diffuse dilatation of the esophagus, yet the credit is due Meltzer, who first pointed out the important relation between the two conditions, and demonstrated clearly that the dilatation is really a stagnation ectasie due to the contracted cardia produced by a disturbance of innervation. On the other hand, Rosenheim maintains that the cardiospasm is secondary to the dilatation. Strauss believes that both the spasm and dilatation occur simultaneously, both due to a vagus paralysis. On the other hand, Fleiner looks upon the dilatation as congenital though it may remain latent for years. Myer points out that this affection is frequently observed in enteroptotic individuals in whom it is not unusual to observe other spastic conditions. There is a general consensus of opinion, however, that cardiaspasm is really the primary state and that the dilatation is the result of this spasm, for by means of the fluoroscope the increasing dilatation has been demonstrated as a result of the spasm. We are firmly convinced, too, that the cardiospasm is the primary condition and that dilatation is a subsequent manifestation. This case, which we have just reported, presents another illustration of how a disturbance of innervation may finally lead to organic changes.

DISCUSSION.

Dr. Baetjer: The patient was not seen by me, but by Dr. Cotton. Dr. Friedenwald has asked me to show the slides. Here are Dr. Cotton's notes, which I will read:

Lantern slides, case B. F. C. Dilatation oesophagus from a long standing cardiospasm.

1. First plate taken; placed as usual for stomach examination. Shows: Large barium shadow above diaphragm—shaped like reversed stomach. Six-hour barium in ileum and caecum stomach not shown.

2. Plate taken higher up (immediate). Shows large barium shadow shaped like reversed stomach above diaphragm and overlapping heart shadow; 24-hour bismuth in colon. Stomach not shown.

3. Same exposure as No. 2 except 7 hours after bismuth meal. Shows barium shadow above smaller and overlapped by heart shadow. Stomach not shown.

4. Right lateral chest exposure (immediate). Shows large conical shaped barium shadow—extending to neck—base of cone downward; shadow of heart and great vessels anterior.

5. Same exposure in No. 4—except taken 3½ hours after bismuth.

6. Plate immediate. Shows the large barium shadow like reversed stomach above diaphragm 12-hour barium in ileum and caecum. Barium shadow in iliac fossa thought to be stomach residue.

7. Plate same as No. 6 and showing same small irregular shadow in left iliac fossa interpreted as stomach residue.

8. Same exposure as Nos. 6-7, but taken one hour later; small diaphragm inadvertently used partly obscures view. Shows the barium shadow above diaphragm and stream of barium extending to quite large dense barium shadows in left iliac fossa in same location as similar smaller shadows in 6 and 7. This plate justifies a diagnosis of dilated oesophagus with gastroptosis and the lower border of stomach being in left iliac fossa.

9. Plate taken 24 hours after swallowing a buck shot attached to a string and 48 hours after barium. Shows small barium residue in dilated oesophagus; shot in left iliac fossa in same location as barium shadows in 6, 7 and 8. This plate confirmed diagnosis made in plate No. 8.

Dr. Jacob Kaufmann, New York City: Similar cases have been seen by me. I wish to report one in particular, because of the good effect of long continued medical treatment. The patient was first observed twelve years ago. He was an undeveloped man, who had never weighed more than one hundred and twenty-five pounds, and who in youth had been constantly under treatment of orthopedists. When I saw him he complained of eructation of food after he had gone to bed and assumed the recumbent position. The food would come up then, and he had to cough it out. In passing a stomach tube I found a most pronounced stagnation of food, as I thought then, in the stomach. It took over two weeks of daily sessions of long continued lavage to remove solid particles of different kinds of food taken before the beginning of the treatment. Large tubes with wide openings were used and had to be taken out frequently to remove solid particles plugging of the openings of the tube. Since the stagnating mass showed a marked bacterial fermentation and great quantities of lactic acid I thought we were dealing with cancer and had the patient operated on. We found a normal stomach. After the operation stagnation of food continued as before and since the button had not passed it was removed at a second operation and a larger opening secured. When the same stagnation continued even then it dawned upon me that the stagnation was located in the oesophagus. I then taught the patient to wash out the esophagus every night before retiring. He has done this practically up to this day. He eats to his heart's content, everything he wants to, and whatever does not go down, he washes out before going to bed. He attends to his business, and I think he is better off than he would be if we had tried to treat his cardiac orifice by surgical methods.

Reference was made to trying to pass the constricted cardia by tubes. I think that this is not always a recommendable procedure. In many cases, the original disturbance is spastic contraction of the cardia. At a former meeting, I pointed out, that the frequent use of ice cold drinks may be a cause for the development of cardiospasm. As an illustration: A young Irish girl came to New York during the hot summer weather, and got to drinking iced drinks. She developed severe epigastric pains and vomiting and had a gastroenterostomy performed because of a diagnosis of gastric ulcer. Since the operation brought no relief she came to the German Hospital where an X-ray showed marked dilatation of the esophagus. She, too, was taught to lavage the oesophagus. [Diagnostic errors of that kind are less liable to happen nowadays with the greatly improved methods of X-ray examinations. Several times I tried to persuade the first patient to have an X-ray taken.]

He claimed that the two operations had been enough for him, and that he was getting along all right with lavage. Lavage benefits not only by removing stagnating and fermenting masses, but also by improving the catarrh, which is invariably associated with the condition; and if you want to relieve the cardiospasm and the stenosis associated with it, I think that atropin is valuable. With this and proper diet, one can get better results than by the attempt to pass the tube through the cardia.

Dr. S. J. Meltzer, New York City: I believe that I have spoken several times before this society on the subject of cardiospasm. Every time when I did so I had to look up that word so as to know it. I have no reason to claim priority for the *term cardiospasm*. That designation was brought forward by Mikulicz in 1881. He was then trying for the first time to use a esophagoscope, and believed to have seen in several cases the constriction of the cardiospasm. It is quite probable that he was mistaken in his observations and their interpretations. The clinical condition which is now quite extensively known and was first described by me and I never called it "cardiospasm," but looked upon it as a *functional stenosis.* I described the first clinical case in 1888. Dr. Einhorn knows that case, and a year later described another case. The functional stenosis is based upon that which I spoke of here a year ago, namely, a Disturbance of the Law of Cross Innervation. The process is as follows: The cardia, as we know, is normally in a state of moderate tonus. With each deglutition the swallowed mass remains resting above the cardia until the moderate contraction of the lower part of the esophagus drives the mass into the stomach. The degree of this contraction need not be very great. It need only be slightly stronger than the degree of the tonus of the cardia in order to overcome it with ease. If then for some reason the lower part of the esophagus loses somewhat of its contracting power then the food will accumulate above the cardia even if its tonus is not increased. In other words in these cases there is no spasm of the cardia at all, but only a diminution of the contractility of the esophagus. These cases look like the present case of Dr. Friedenwald; the dilatation does not have a spindle but a funnel shape form, indicating the place at which the lower part of the esophagus starts to give way. Of course there might be cases in which the tonus of the cardia is really increased, the nature of the increase may be merely functional or it may be due to a small erosion in the cardia; such cases may be indeed termed cardiospasm.

I wish further to emphasize a point which I brought out many times before, namely that to my mind it is a very simple thing to diagnose functional stenosis or cardiospasm. We need only to listen to the deglutition sounds; if you find the pressing sound (or second sound) permanently absent you may be sure that the case is a stenosis of the above mentioned type. In any other condition in cancer, for instance, you can always hear a weak deglutition sound indicating that some of the food is passing into the stomach. Finally I wish to refer to a point which I brought out in my first paper on the subject. I described then the method which my patient, who was suffering from the disease in question for about nineteen years, has developed, by which the whole mass resting above the cardia can be brought into the stomach at once. I shall not enter here into the details of the method which she used, and which I believe many can be brought to use it. When this mass is successfully brought into the stomach *a sound is produced which can be heard three or four yards away from the patient.*

DR. CHARLES G. STOCKTON, Buffalo, N. Y.: I should like to call attention to a fact that some here will remember: that, twenty years ago, the late Dr. Kinnicutt presented before the Association of

American Physicians a case similar to that shown by Dr. Friedenwald. I think that was before the days of the X-ray.

I am somewhat perplexed, in hearing the discussion today, to find that attention has not been directed to what I thought we had settled upon, years ago, as the proper procedure to be followed in these cases, and to the almost universal success following that procedure, viz.: dilatation or over-distention of the contracted point. That remark is brought forth by what Dr. Kaufmann said about medical treatment being desirable in these cases. I do not understand what he means by that.

I am also perplexed to know why Dr. Smithies hoped to be able to present the post-mortem specimen of the case he mentioned. I do not quite understand why there should be a post-mortem specimen of the case; it should at least lose much of its pathology.

Dr. John A. Lichty, Pittsburgh, Pa.: I have just a word to say, which may defeat Dr. Smithies' end, and may be of some degree of encouragement to Dr. Friedenwald. I have under my care at present a case of cardiospasm, and have struck upon a course of treatment that I have not found described heretofore.

In this case, we made the diagnosis of cardiospasm with the X-ray. I asked Dr. Chevalier Jackson to look into the esophagus to determine whether after all the condition might not be malignant. He said that he could pass the Einhorn button through the spastic cardia, which he did. This was on the 26th of January. The man immediately afterwards began to feed himself. We got 2700 calories through each day, and the patient began to pick up in weight. He handled the feeding entirely himself. After six weeks of feeding of this kind, I took an ordinary stomach-tube and slipped it down along the side of the Einhorn tube, and it went into the stomach without difficulty. The patient is now eating food on the side of the tube, even though he has the Einhorn tube still in. I believe that this man is going to find that he is entirely well, after we remove the Einhorn tube. The method of dilatation, so well described by the late Dr. Meyer, of St. Louis, and the method to which Dr. Stockton referred, is very disagreeable. The food will irritate the injured or traumatized part so that you cannot dilate again for eight to ten days. I do not believe that we accomplish much in that way; but with this method, in which we can feed the patient and keep his nutrition up, I believe we can overcome the trouble in the treatment of cardiospasm. I wonder what would have happened in Dr. Friedenwald's case, if, with the esophagoscope in position, the Einhorn tube could have been slipped through and the patient fed with the tube in place.

Dr. ————: Is the tube in the stomach?

Dr. Lichty: Yes, it is in the stomach. We did not allow it to go into the duodenum. We have examined it with the X-ray, and the button is there now. This is a very pronounced case—the oesophagus is equally large as the case of the specimens of which were exhibited. The man had lost forty pounds in weight, and has now regained it.

DR. FRIEDENWALD, closing: The great difficulty in passing through the strictured area is great. I do not quite agree with Dr. Kaufmann in his statement that we should treat these cases medically. That is quite true in some instances of cardiospasm without dilatation of the esophagus, but when there is a dilatation of the esophagus at hand, the stricture must be forcibly dilated. When the stricture is not impermeable, however, it is possible to dilate with the Meyer dilator, but in an impermeable stricture such as we had in our case, this is not possible.

The only method left open in these cases is to have the patient swallow a string which is used as a guide over a perforated bougie. This is the method that Dr. Smithies has employed with success. I have used it, too, and believe that it should always be applied in these cases.

DR. FRANK SMITHIES, Chicago, Ill.: I have enjoyed hearing Dr. Friedenwald's case particularly because he did an autopsy. Those who visited my clinic during the past year have seen a man who is supposed to have a "thoracic" stomach. I have two lantern slides showing his esophagus. He was a member of the first baseball team that invaded Cuba. This man, seventeen and a half years ago, had a gastrostomy performed on account of obstruction of the cardia. After that, he was treated by the passing of large tubes up from the stomach and out through the mouth. This improved him. His gastrostomy wound healed, and he was able to swallow. Then, for a year and a half, bougies were used. Sixteen and a half years afterwards, he came under our observation, and we found that he had a positive Wassermann test. This is the cardiac shadow taken last week. Our attempt to fill the esophagus proved a failure. We put a stomach-tube in, and poured in 2800 c.c. of barium mixture in an attempt to fill the sac, but then stopped because the patient was getting into a state that we thought he would die from cardiac failure. I hope next year to present the autopsy. (Laughter.)

MAGNESIUM SULPHATE SOLUTIONS IN THE TREATMENT OF SPASTIC CONTRACTURES OF THE RECTUM AND SIGMOID COLON.

By Horace W. Soper,

ST. LOUIS.

This paper deals with the action of magnesium sulphate upon the intestinal tract. Meltzer, in his experimental work on animals, found that solutions of magnesium sulphate whether given intravenously or applied directly to the mucosa of the intestine produced inhibition of peristalsis. The deduction follows that solutions of the salt applied locally should have a tendency to overcome hypertonic contracture of a section of the intestine.

Two thousand cases of chronic constipation were studied, showing that contractures in the distal half of the colon were present in a large percentage of the cases.

Two hundred and forty cases were treated by direct applications of magnesium sulphate solution to the contracted segment through the sigmoidoscopic tube. The results were very satisfactory, many obstinate cases are apparently restored to a condition of normal colonic function.

Enemata of three ounces of the saturated solution of magnesium sulphate were efficacious in the treatment of postoperative abdominal distension.

One year ago, Meltzer in the symposium on gall-stones before this Association, sugested the employment of a 25 per cent. solution of magnesium sulphate, given through the duodenal tube, in certain obstructions of the common duct, hoping thereby to produce a relaxation of the circular muscle fibers of the ampulla of Vater. The experimental work of Meltzer demonstrated that solutions of magnesium sulphate produced an inhibitory influence upon the peristaltic movements of the intestinal tract of animals whether the salt was given intravenously or applied directly to the mucosa. Briefly his hypothesis is that the phenomena of peristalsis consists in a contraction of the proximal and relaxation of the distal part of a section of the intestine, following the law which he designates as "contrary innervation." The purgative action of magnesium sulphate when given by mouth results from the fact that a part of it acts by being converted into sodium sulphate and magnesium carbonate, substances which increase the irritability of the exciting or contractile phase of peristalsis, while the remaining unconverted magnesium sulphate acts by increasing the inhibitory or relaxing phase of peristalsis. From this hypothesis we may deduce that a condition of hypertonus in a section of the intestine is due to a disturbance of the law of contrary innervation, *i. e.*, a preponderance of stimulation occurs in the exciting or contractile phase.

For a long time I have been interested in the muscular contractures which occur in the lower colon and rectum as revealed by sigmoidoscopy. Furthermore, I am convinced that these contractures are pathological in character and that they are etiologic factors in many cases of chronic constipation.

The deduction is therefore obvious that solutions of magnesium sulphate applied directly to a hypertonic or contracted segment of the bowel should have the effect of producing a relaxation. In a series of cases I have demonstrated that magnesium sulphate is really capable of overcoming such contractures and that this action may be utilized in the treatment of obstinate constipation.

In a review of 2,000 cases of chronic constipation taken from my office files (dating from 1909) the following classification is tabulated:

Nine hundred and sixty cases of spastic constipation, the spasticities being located in the rectum, sigmoid and descending colon.

210 cases in which the entire colon appeared to be in a state of hypertonic contracture.

406 cases of chronic atonic constipation.

424 cases recorded as "indeterminate," meaning thereby that neither contractures nor atonic conditions could be ascribed as etiologic factors.

Cases of partial intestinal obstruction due to organic conditions such as strictures, growths, inflammatory processes, etc., are not included in the above series.

The procto-sigmoidoscope is the most valuable diagnostic agent in the detection of hypertonic contractures. No one experienced in its use will doubt the existence of the spastic form of constipation.

In cases in which the contracture is beyond the reach of the sigmoidoscope the diagnosis is made by the subjective symptoms, the characteristic fragmentary feces, combined with a strongly contracted palpably tender descending colon or sigmoid loop.

The X-ray diagnosis of contractures is extremely unreliable, chiefly because of the position and form of the sigmoid colon. The bismuth or barium enema will pass through an organic or spasmodic stenosis whose lumen is of very small calibre. It may canalize contracted areas which are concealed by the distended portion of the bowel and thus escape detection, especially as palpation is practically impossible in this region. Moreover, the bismuth or barium meal given by mouth is frequently scattered in a thin column along the lower colon, giving an impression of contracture when no narrowing exists. Also the degree of tonus frequently changes in the normal colon and interpretation presents extreme difficulties. The illustrations show the difficulties encountered in attempting to make Roentgenoscopic diagnoses of contractures.

Contractures of the rectum and sigmoid vary in intensity from mild contractures which readily yield to dilatation to strong spasms which form unyielding obstruction. The contractures may be located at any point in the rectum, sigmoid, colon or descending colon including the splenic flexure. The most frequent site is the recto-sigmoid junction. Severe contractures at this point present a syndrome which I have designated "sigmoidospasm."

An obstinate form of constipation results from combined contractures and dilatations, *e. g.* (a) atony of rectum and sigmoid associated with contracture at descending colon; (b) contraction at recto-sigmoid angle and atony of sigmoid loop; (c) contracture at splenic flexure and descending colon and atony of cecum.

Action of Magnesium Sulphate Applied Directly to Contractures. Observation Made Upon 240 Cases.

1. A saturated solution was found to be most efficient.
2. The solution was applied by means of cotton applicators through the sigmoidoscopic tube (knee-chest position). A characteristic diffuse, deep pink color of the mucosa is observed within from 10-20 seconds.
3. Mild contractures disappear within a few seconds. Mod-

erate contractures require a minute or two. Strong spasms require a series of applications to produce relaxation.

Treatment of Constipation Exclusively by Saturated Solutions of Magnesium Sulphate.

Eighty obstinate cases which had been under my care for a year or more. They had been under treatment by dietetic measures, enemata of oil, agar-agar, dilatations by means of oil applicators, insufflations of air, etc. None of them had been able to get along without the use of the oil enema. Sixty-eight cases have apparently been completely restored to a condition of normal colonic function. The number of treatments required in these cases varied from ten to thirty applications given every second or third day.

Five cases were complicated by inflammatory induration and distortion of the bowel at the recto-sigmoid junction, consequently treatment was ineffectual.

In seven cases the contractures were overcome, but normal colonic function was not restored.

Seventy-two new cases of obstinate spastic constipation, *i. e.*, cases that were of long standing and that had not been previously under my care. Sixty-four cases were successfully treated with satisfactory restoration of bowel function. Eight cases were extremely neurotic individuals, who could not stand the treatment, although no real pain is produced by it.

Sixty-eight cases of obstinate spastic constipation, cases of long standing constipation, but which presented contractures that were moderate in character, *i. e.*, that readily responded to treatment, and required but from 6 to 10 applications to restore good colonic function.

The plan usually adopted is for the patient to return for treatment every second day, gradually lengthening the interval between treatments as the bowel function is restored. Purgatives and enemata of water must be avoided inasmuch as they both increase the tendency to contraction. Oil enemata may be used in conjunction with the magnesium sulphate treatment, when it is not convenient for the patient to come to the office for frequent treatment.

Cases in which the contracture is beyond the reach of the sigmoidoscope are treated by passing a soft rubber catheter through the sigmoid tube and injecting two ounces of the magnesium sulphate solution, the patient retaining the knee-chest position for several minutes thereafter.

Sigmoidoscopic examinations of patients in whom normal function had been restored revealed the absence of previously demonstrated spastic obstructions.

While it is too early to assert that these patients are permanently cured, nevertheless some of the most obstinate cases have remained in a satisfactory condition for more than ten months.

The Action of Enemata of Magnesium Sulphate in the Relief of Postoperative Abdominal Distensions and Gas Pains.

Observations of the condition presented by postoperative abdominal distension suggested the use of magnesium sulphate enema as means of relief. X-ray examination of six cases revealed that the colon alone was involved, no gas being found in the small intestine. The rectum and lower sigmoid were free from gas. Sigmoidoscopic examination showed the presence of marked contractures in the rectum and lower sigmoid. In a series of 30 cases I used an enema of saturated solution of magnesium sulphate given through the rectal tube. The technique is as follows:

*Three ounces of saturated solution of magnesium sulphate is introduced by means of the rectal tube passed four or five inches into the bowel, elevating the hips whenever possible, and having it retained as long as possible. This procedure may be repeated every day, or several times a day if so desired.

The effect was quite remarkable. Large quantities of flatus were expelled, followed by immediate relief in twenty-four of the cases. The enema is usually retained for not more than five minutes. Occasionally it is retained for 24 hours; however, no harm results, as no toxic effects have been noted. Defecation often follows, not characteristic of a purgative dejection, but should the colon happen to contain much fecal matter, many passages may result. No rectal pain nor tenesmus follows the daily use of the enema. No evidence of griping or increased peristalsis occurs. The magnesium sulphate solution evidently merely relaxes the contracted rectum and lower sigmoid and permits the retained gases and fecal matter to pass out of the distended colon.

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*Meltzer warns against giving too large a dose as toxic effects have been observed particularly in animal experimentation.

The enema was also used in two cases of partial obstruction of the lower colon and sigmoid with very beneficial results. One such case was seen in consultation with Dr. H. G. Mudd. Patient, female, age 75 years, presented the clinical picture of acute intestinal obstruction with great distension of the abdomen and much pain. Operative intervention was seriously considered. Patient had previously received large doses of cathartics, enemata of water, etc., without effect. The magnesium sulphate enema repeated from time to time was very effective. A later study of the case revealed that an adhesive periproctitis had resulted in a partial obstruction in the upper rectum. Evidently a muscular spasm was partly responsible for the stenosis which was overcome by the magnesium sulphate.

A second case was seen in consultation with Dr. John R. Caulk. The obstruction was located in the sigmoid loop, due to an extension of a pelvic abscess. Magnesium sulphate was again very effective, the patient making a good recovery. Within a week the operation of prostatectomy was successfully performed.

Realizing that personal enthusiasm is apt to impair one's judgment in forming an opinion as to the value of a therapeutic procedure, I asked several surgeons to try the enema in cases of postoperative gas pains and report their results to me. The replies representing 222 cases are summarized as follows:

Dr. Francis Reder: Magnesium sulphate inferior to alum enema combined with pituitrin.

Dr. W. H. Vogt: Magnesium sulphate inferior to intramuscular injection of pituitrin.

Dr. N. B. Carson: Used dilute solutions and results not satisfactory.

Dr. Mary H. McLean: Favorably impressed.
Dr. V. P. Blair: Favorably impressed.
Dr. Ernest Jonas: Favorably impressed.
Dr. G. D. Roylston: Favorably impressed.
Dr. Willard Bartlett: Treatment of great value.
Dr. E. J. Neville: Treatment of great value.
Dr. O. H. Elbrecht: Treatment of great value.
Dr. John McH. Dean: Treatment of great value.
Dr. John R. Caulk: Treatment of great value.
Dr. H. G. Mudd: Treatment of great value.
Dr. P. Y. Tupper: Treatment of great value.

Dr. H. L. Nietert: Used it for years, and finds it of great value.

Dr. Major G. Seeling: Used it in 50 per cent. solution combined with turpentine and green soap.

Dr. Tupper thus describes its effect: "Within a short time, ordinarily within one-half hour, there has been a free passage of flatus accompanied, not infrequently, with a fecal evacuation. Apparently the salt acts directly upon the rectum and lower sigmoid causing relaxation of the muscular coat without producing peristalsis of either the large or the small intestine. Because of this the therapeutic measure is safe and especially serviceable soon after operations upon the intestine where evacuation of flatus is necessary for the patient's comfort and safety and where active peristalsis might produce serious results.

Magnesium sulphate thus applied apparently is more effective than the enemata ordinarily used and is apparently free from the discomfort and danger accompanying their use."

Conclusions.

1. Spastic contractures of the lower colon are etiologic factors in many cases of chronic constipation.
2. These contractures are the result of disturbances in Meltzer's law of contrary innervation.
3. A saturated solution of magnesium sulphate applied locally to the contracted segment produces a relaxation. Repeated applications finally overcome the spasticity and permit the restoration of normal colonic function.
4. Contractures in the rectum and lower sigmoid with accompanying dilatation of the colon are found in many cases of postoperative abdominal distension. Magnesium sulphate enemata are very efficacious in relaxing the contractures and thereby relieving the distension and "gas pains."
5. Enemata of magnesium sulphate are also very useful in partial organic obstructions in the rectum and lower colon, inasmuch as they relax accompanying muscular contractures without stimulating peristalsis.
6. Magnesium sulphate solution applied by means of the cotton applicator greatly facilitates the introduction of the sigmoidoscope.

REFERENCES.

[1] S. J. Meltzer: "The Relation of the Purgative Action of Magnesium Sulphate to Peristalsis, and the General Law of Crossed Innervation." (*Archiv. Internal Medicine*, June, 1915, Vol. 15, No. 6, page 955.)

[2] H. W. Soper: "Sigmoidoscopy." *International Clinics*, Vol. IV, 1916, page 260.

DISCUSSIONS.

Dr. John A. Lichty, Pittsburgh: After hearing Dr. Meltzer's paper at Washington, last May, it occurred to me that it would be well to adopt his principle in certain gall bladder cases. I have had a number of cases during the past year, but I had not intended to speak of them, because I had nothing definite to say.

There are two classes of cases in which this method might be applicable. One consists of cases of jaundice, in which we do not have a definite history of gall stones, but simply a cholecystitis or inflammation of the common duct. Probably in these cases it would favor relaxation and drainage of the common duct. Another class of cases is those of gall stone, in which an operation is contraindicated on account of cardiac or renal conditions, or other conditions that may make it hazardous. In these two classes, therefore, we institute the principle suggested by Dr. Meltzer. We cannot say anything definite. We feel that the jaundice will disappear more rapidly and promptly with this treatment than with any other method that we have attempted. This, of course, is relatively speaking. In the cases of gall-stone, in which operation seems to be contraindicated, it appeared to us that the attacks did not return so soon. The patients seemed to get over the attacks on much less than the amount of medication we had had to employ heretofore.

I am greatly interested in the work which Dr. Soper has done. While it looks like a reasonable thing to do, I can hardly understand how the twenty or more applications of magnesium sulphate are going to give permanent results in spastic constipation. I doubt whether after the treatment was stopped, the constipation would not return just as it had been before.

Dr. S. J. Meltzer, New York City: I have not heard the entire paper, but Dr. Soper was kind enough to tell me a good deal about its contents. I am, quite naturally, delighted with the results. I suggested at our annual meeting last year to introduce magnesium sulphate into the duodenum through the duodenal tube for the purpose of relaxing the sphincter of the common duct. If I am not mistaken this was carried out indeed with some good results. While the fact may be satisfactory, it does not necessarily support my view of the action of magnesium sulphate; because many will simply say: Magnesium sulphate is a cathartic and a patient suffering from jaundice will get better by such a remedy. But my interest is mainly in the assumption that magnesium salt causes a relaxation and the ocular observations of Dr. Soper support directly this view; he has observed the relaxation of the rectum or the colon by the local application of a saturated solution of magnesium sulphate. For hundreds of years it was known that magnesium sulphate acts as a cathartic; but it was generally assumed that this action is due to an increased motor activity produced by the magnesium salt. Since nearly two decades I defend another view, namely, that the main effect of magnesium salts is inhibitory in character and the various effects it produces is by its relaxing power. If you will permit me I would like to tell you the story how I came about to assume this view.

Some nineteen years ago I made in the private laboratory in my own house, all sorts of experiments. Among them I studied the effect of injections of salts into the brain of rabbits. Various salts produced convulsive effects simulating tetanus. But one series of rabbits in which I injected in each case not more than two drops of a 5% solution of magnesium sulphate, the animals turned over on their side and slept for several hours without any sign of convulsions. Since 1880 I am an *inhibitionist*, that is, I entertain the idea based upon various experimentations that all functions of the body are complicated by inhibitory phenomena. Now when I saw the effect of the magnesium sulphate the inhibitory theory made its appearance. I asked myself why physiologists and pharmacologists studied extensively the general and local actions of sodium, potassium and calcium salt while the action of magnesium was practically neglected. It occurred to me that magnesium, perhaps, represents the inhibitory part in the functions of the body and that every investigator was looking only for substances which may cause *motor action*, convulsions, etc., but magnesium does nothing of that kind, it merely *inhibits*. At the Annual Meeting of the Physiological Society in 1898 I have demonstrated three rabbits; two had injections of sodium sulphate and sodium chloride and were manifesting motor effects, while the third rabbit which received magnesium sulphate was lying quietly like being absolutely paralyzed. When I was then asked about my explanation I said that I am not yet ready to state my theory; I merely wished to demonstrate the fact. Since 1905 Auer and I published a great many communications which all demonstrate that magnesium salts exert an extensive inhibitory effect, centrally and peripherally. On animals I have shown that the local application of a solution of magnesium salt upon the inner side of the lumen of the intestines is capable of causing a paralysis of the intestines. Dr. Soper told us that he has observed a similar effect upon the rectum of the human being. It is instructive to state that such a vague theory which entered into my mind a long time ago, bore practical results in the present horrible war. Only recently Dr. Pribram stated that in the extensive cases of tetanus encountered by the central belligerents in the years 1914 and 1915 the use of magnesium sulphate proved to be the most practical means.

I wish to add two remarks. One is the possible suggestion why Dr. Soper obtained *permanent* results. The conditions in Dr. Soper's cases may have been maintained by some *vicious circle;* the cutting through of such a circle may have caused a permanent cure. My second point is, to bear in mind that too large a dose of magnesium sulphate may prove poisonous. In striving to contribute to medical knowledge in some fundamental way I wish pre-eminently that my contributions shall not lead to unfavorable results.

I am aware that in my discussion I have overstepped any sensible time limit and I thank you, Mr. President, and gentlemen, for your indulgence with me.

DR. G. W. MCCASKEY, Fort Wayne, Ind.: It is very gratifying to breathe the atmosphere in which progressive, scientific medicine is in the making. That is what we are doing, and we are honored by the presence and work of such a man as Dr. Meltzer. While he was speaking, some of my experience was going through my head; and I think I can reach an explanation of some things that I could not understand before. For years, I have been used to injecting four ounces of a saturated solution of magnesium sulphate, when other measures had failed to produce proper action of the bowels; and in a large proportion of cases it acted like magic. But in certain other cases it failed. In the cases in which it succeeded we possibly had a spastic condition, and in the others, the opposite condition. If we got results at all, it was only by an osmotic process.

I wish to express my thanks to Dr. Soper and Dr. Meltzer for this work. I think that it offers an explanation of the contradictory experiences that I have often had in the use of magnesium sulphate.

DR. HENRY W. BETTMANN, Cincinnati, Ohio: I think that we make a mistake, if we talk of spasticity of the colon as an entity and if we cling to the old classification of spastic and atonic constipation. There is nothing distinctive about spastic constipation. In every chronic constipation, there are symptoms that suggest spasticity. Spasticity occurs in every form of chronic constipation, and no one can set aside one group as spastic and another as atonic. To pick out one feature of chronic constipation, viz.: spasm of the descending colon and make it stand for a class is not very logical. Spasm is only one particular symptom; and to treat it as a local process is not a scientific way of treating constipation. What causes spasm, we do not know. I am sorry that Dr. Carlson is not here to tell us of some work that he has done on the innervation of the colon. There is reason to believe that certain parts of Auerbach's plexus are the centers for the origination of peristalsis in the colon. The complete innervation of the movements of the colon is not yet fully understood. The X-ray does not reveal the slight peristaltic contractions that are responsible for the onward progression of the colon contents. The mass movements have been seen, but the stimuli that liberate these reflexes are not understood. The taking of food into the stomach liberates a reflex that causes the food to go forward, the so-called gastro-colic reflex. The treatment of constipation cannot be the treatment of an ephemeral spasm. There are different types of spasm in the stomach, which can be seen by the fluoroscope; and these are due to nervous and other causes. To pick out the spasm of the colon as something to treat is not the right way to approach the treatment of constipation.

Dr. Charles D. Aaron, Detroit, Mich.: I should just like to add that I have introduced 100 milliliters of a saturated solution of magnesium sulphate into the duodenum a great many times, but it was first diluted in one liter of water. I have even used 120 mils. of a saturated solution in this way. That would mean magnesium sulphate itself to within a small fraction of the full volume of the solution; for it is very soluble. Never but once have I failed to add the diluent. Once I did introduce 100 mils. of a saturated solution, without dilution through the duodenal tube, and without any bad effect; and I am frank to admit that I did not then know that magnesium sulphate could act as a poison, as Dr. Meltzer has told us today. If I had known that, I should not have taken the chance, but I have so many times administered full doses diluted that I can say you need not hesitate to use 100 mils. of a saturated solution in a liter of water, introduced through the duodenal tube, in the case of an adult. Of course it would produce a copious movement within one or two hours, and the patient would eventually have two or three, or perhaps five of six movements, without pain or discomfort. I have given magnesium sulphate intraduodenally in so-called intestinal stasis—for instance, when the patient has been told by the surgeon that he must undergo a surgical operation. I was very much interested in what Dr. Meltzer said, because he led me to reason out why magnesium sulphate does good. Of course we all know, as Dr. Bettmann has said, that spastic constipation is really not an entity. In all probability it is due to vagotonia. If this be true, and Dr. Meltzer tells us that magnesium sulphate inhibits, I think the explanation of the effect of magnesium sulphate in Dr. Soper's cases is clear.

Dr. J. C. Hemmeter, Baltimore: The movements of the colon are controlled by extrinsic and intrinsic nerves. These salts have an effect when the colon is cut off from all extrinsic nerves. If you look up the reports of the work of John Bruce MacCalloum executed at the University of California in 1906, you will find the explanation of the effect of electrolytes on the movements of the intestines. The intestinal peristalsis is caused by barium salts, even painted on the outside of the intestines. When the intestines are removed from the body, they will move when $BaCl_2$ Na_3 PO_4 Na_2 So_4 magnesium sulphate is painted on the outside, and the movement will be arrested when calcium chloride is painted on the outside. You are really dealing, here, with the intrinsic nerve ganglia of Meissner and Auerbach or the muscle itself. The colon is supplied by nerves that run partly with the sympathetic system along the colonic and inferior mesenteric nerves and partly pelvic visceral nervi erigentes. The effect of the salts has nothing to do with the vagus, in my opinion, because the colon is no anatomic connection with the vagus and ileo ebcause these peristaltic effects of salts will occur even after the colon is removed from the body.

Dr. Soper: The question of Dr. Bettman's as to whether or not there is such a thing as spastic constipation, can only be settled by men doing sigmoidoscopic work. The X-ray is unreliable inasmuch as the loop of the sigmoid is so located as to be inaccessible to palpation in the majority of cases. Furthermore a distended portion of the bowel may overlap a ocntracted area and the X-ray plate will not show the contraction. However, one watching these cases with the sigmoidoscope will soon become convinced that there is such a thing as spastic conditions of the sigmoid, that these spasticities are not ephemral, but definite spasms. Spastic constipation is really a clinical entity. Some of the points in establishing a diagnosis are as follows: (1) the history of obstinate constipation, (2) the characteristic, fragmentary feces, (3) the pain and discomfort produced by cathartics and enemas, (4) the discovery of a definite spasm by means of the sigmoidoscope, a spasm which is impossible to overcome by any sort of a tube, (5) verification that the spasm is not of organic nature by being able to finally overcome it by means of magnesium sulphate applied locally. The question as to why the result of treatment, is usually permanent can be answered in this way: Normally, the operation of the law of crossed innervation produces certain movements which we designate as normal peristalsis. These spasticities in the sigmoid appear to act as obstructions to this normal peristalsis. If they are removed by means of applications of magnesium sulphate solutions, the law of crossed innervation can operate in the normal way, thus restoring a normal colonic function.

CANCER OF THE RECTUM.*

By Jerome M. Lynch, M. D., M. R. C., U. S. A., Fellow of the American College of Surgeons.

Cancer of the rectum and colon has increased one hundred per cent. in the past fifteen years, according to the report of Vital Statistics of the Registration Area of the United States. Bloodgood has well said that in the life history of every malignant growth there has been a moment when it was surgically curable and the lesions under consideration offer no exception to this admirable axiom. To recognize and to seize upon this precious moment is no less than to control the life destinies of the affected individual.

Results of Surgical Treatment.—Of 491 cases studied we have operated upon 335. The hospital mortality was sixteen per cent. Forty-one have lived one year; forty-five two years; thirty-three three years; twenty-two four years; twenty-six five years and seventeen six years.

Patients not replying to circular letter have been classified as dead when last heard from. As in all clinics situated in cosmopolitan centers where the population is in constant flux it is impossible to follow a large number of the patients. Thus the statistics as above created are necessarily less favorable than if every case had been followed to date.

Whatever disability exists as results of operation does not interfere with livelihood gaining. One of our patients who was bankrupt when his rectum and sphincter were removed, and who leaks at times, now makes four trips to Europe and has earned over a million dollars since operation. We cannot overemphasize the plain fact that postoperative conditions, no matter how unfavorable as to function, do not interfere with the usefulness or economic independence of the patient.

Incontinence is a relative term. Its importance has been grossly exaggerated. As no horse is sound, so no human body is without defect and even great defects are compensated for by the natural endowment of the individual to meet such obligations. It is simply a question of getting used to the discomforts of a colostomy or a leaking anus—a psychological phenomenon well worthy of consideration. Think of the innumerable women torn in childbirth who have been incontinent for a quarter of a century, yet who efficiently and without affront to their families perform their daily work. Because of this psychological element we strongly prefer to have a relatively incontinent normal anus rather than upon the abdomen.

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*Read before the American Gastro-Enterological Association, Atlantic City, May, 1917.

This briefly is the result of surgical therapeusis in our series. What stronger argument could there be for discussing the diagnosis and the indications. These statistics show that rectal cancer operated upon even after great delay and by poor methods is not hopeless. If with these limitations we get results, how important, therefore, is the early diagnosis and how promising the outlook for the future.

What have been the methods of study in this series of 491 cases? Of first importance is a flat contradiction of some still prevailing convictions, to-wit: That the operation is hopeless; that the cancer patient is cachetic or has lost weight; that age is of importance; that pain is a prominent symptom and that a tumor can always be felt. The very occurrence of these symptoms spells inoperability.

What are the important symptoms from the modern standpoint in order of diagnostic and therapeutic importance?

First.—Constipation.—This we believe to be the very first and earliest of all symptoms. It is undoubtedly protective in type, being perhaps the result of biologic reaction to the influence of the new growth. There are, however, several hypotheses as to its origin depending upon the path of inhibitory transmission rather than upon its origin or occurrence. Of the latter and of its protective nature there can be no doubt. Certainly it is not due to mechanical obstruction of the growth.

Second.—Stomach Symptoms.—We have repeatedly referred to these as esoteric as contrasted to hemorrhage and the like, which are exoteric. Chronic indigestion, so frequent a sign of peripheral pathology, is just as significant of rectal cancer as of a chronic appendix.

Third.—Blood or Bloody Stools.—This is usually the first exoteric sign. It can occur without ulceration, in which case it may be due to a blocking of the return circulation in the valveless veins leading to the liver. In any event hemorrhage so commonly associated with cancer (10 per cent. of our cases of 491 had been operated upon previously to our seeing them, for hemorrhoids) is a frequent source of the blood. In a large proportion of the cases, however, it is due to ulceration.

Fourth.—Frequent and Imperative Desire to Move the Bowels Followed by Explosive Discharges of Gas, Blood and Mucous.—This symptom is usually spoken of as the diarrhea of cancer. It is not in reality a diarrhea in that feces are rarely passed.

These are the classical symptoms which every gastroenterologist should know. Other symptoms occasionally noted are an indefinite pelvic discomfort and pain or tenderness over the cecum which was mistaken for right-sided pathology.

Diagnosis.—A patient presenting any one of the above symptoms should have a rectal and proctoscopic examination as a matter of routine. In our series of 491 cases, fifty-six per cent. of the tumors were within 7.5 cm. of the anus; sixty-nine per cent. were within 10 cm. of the anus and thirty-one per cent. were oral to this. It is quite evident, therefore, that more than a half were within reach of the finger, that two-thirds could have been diagnosed under anesthesia by the finger and that all except the sporadic cases in the colon could have been diagnosed by the proctoscope.

Duration of symptoms in this series was eight months. During this period many of the cardinal diagnostic symptoms already referred to had been present, so that at any time a diagnosis could have been made had the patient been examined.

Age.—In our series of 491 cases, four per cent. were under thirty years of age; seven per cent. thirty-five. According to the United States Bureau of Vital Statistics one-half per cent. of cases of rectal and colonic cancer were in children under nine years of age, two and seventy-five one-hundredths per cent. under nineteen years, proving that cancer is not confined to any age, and that while it occurs more frequently in middle life, still for all we must recognize the danger of placing too much importance on age.

A word must be said regarding the pernicious habit of biopsy for diagnosis. At the Symposium on Inoperable Cancer of the New York Academy of Medicine, Dr. Robt. Abbe remarked that in the treatment of carcinoma by radium, the biopsy wound itself was one of the last to heal and was very stubborn.

Treatment.—Operability: In our series of 491 cases extending over a period of nineteen years one hundred and fifty-three were considered inoperable. Of great importance is the history of the advance of our technique and a more liberal understanding of the possibilities. From a study of unexpected results in many so-called inoperable cases we are convinced that even in the late cases, except when the peritoneum is involved, there is always a fighting chance. Of the one hundred and fifty-three cases considered inoperable none have been so classified because of the extent of involvement in the rectum itself.

Our operability for the total number is sixty per cent. This high percentage is due to the fact that Dr. Tuttle kept no record of inoperable cases. By operability is understood radical extirpation of the growth. In the past five years our operability has risen to seventy-four per cent. Let it be clearly understood that this refers to growths strictly localized in the rectum. As to the indications for radical treatment, when adjacent organs are involved our statistics show that we have often removed a part of the vagina, a part or whole of the prostate, seminal vesicles, urethra and uterus, several coils of intestine and part of the bladder. In many instances it is necessary to perform an exploratory laparotomy to determine whether the growth is operable.

Choice of Operation.—Operable cases: 1. Combined. 2. Perineal. 3. Abdominal.

We have performed the combined operation 111 times; in 36 of these cases it was performed in two stages. It is our operation of choice.

The perineal has been performed one hundred and two times; the abdominal twenty. Formerly we used the following operations now in disuse: Kraske, twenty; modified bone flap, thirty-two, and intra-rectal eighteen. When possible, for the psychical reasons already described, we always place the anus at the normal site. We prefer to perform the operation in one stage if it is possible, but, if necessary, we divide it into two stages.

The peritoneal operation is our operation of choice in very fat or in extremely debilitated people. In all cases, as a matter of routine, we always remove the coccyx. Preliminary colostomy is always done when the growth is within two cm. of the anus in order to prepare for the extensive removal en bloc necessitated by lymphatic involvement.

We have for some time abandoned rectal resection for the reason that in all of these cases the operation was followed by stricture. This is due to the presence of a terminal blood supply in the rectum rather than, as commonly supposed, to the absence of peritoneum. Exceptional work on dogs has been done by Barber which has confirmed us in this belief. It is the ischemic rather than the peritoneal denudation that produces the stricture. It is axiomatic that the amount of scar tissue is in reverse ratio to the blood supply. This important contribution will form the basis of a subsequent paper.

Palliative Operative Treatment.—What can be done in this type of case is still of great importance. Until earlier diagnoses are made many cases will continue to fall in this class. If this paper served no other purpose than to convince the profession of the necessity of early colostomy in inoperable carcinoma of the rectum, it will have done some good. The fixed attitude toward colostomy is that it should be postponed until obstruction supervenes. This is certainly not in accord with the facts as we find them in thirty-six cases for cancer alone and in over one hundred for other conditions. It can be done under local anesthetic.

What are the advantages of early colostomy, in inoperable cancer as opposed to the supposed disadvantages? It reduces the inflammation often converting an inoperable into an operable case. It obviates intestinal obstruction and its accompanying symptoms of pain, constant secretion and defecation, permits rest and sleep and insures recuperation. The patient renews his normal routine as to habits and diet. It stops hemorrhage. In short, it places the parts at surgical rest. If early it is without notable mortality; if late this rises to forty or even fifty per cent.

Local Cauterization.—This is frequently of great value; it stops pain and limts secretion and odor. If frequently repeated it may keep a patient alive for many months.

Treatment by radio active substances, fulguration and by biochemical derivatives is not here considered.

CONCLUSIONS.

1. We would urge that digital and prostoscopic examinations be made routine in all patients presenting gastro or intestinal symptoms. If this is adapted a great many cases will be diagnosed early and saved.

2. That all cancer cases should be referred to a surgeon, as he is best fitted to pass judgment as to whether they are suitable for operation or not.

3. If operable, colostomy should be performed as soon as possible, thereby saving much suffering and discomfort.

4. That no patient should be denied a radical operation until it is proved beyond doubt that it is not justifiable.

5. That our technique is now more perfect and consequently we are saving many cases which previously died from shock and peritonitis.

DISCUSSIONS.

Dr. Horace W. Soper, St. Louis: In X-ray examination of the lower colon and sigmoid we not only often miss the growth that is present, but frequently, in our experience, apparent growths have been found when in reality they were not there. Dr. Lynch's term, segmental infection, is a very happy one. It is often located at the recto sigmoid angle. Contractions here are very frequent, and are often of very long duration. After dilatation of this area of the bowel, the segment is found to be undergoing a lowgrade inflammatory process. The mucosa is frequently thickened. Pus organisms can often be isolated. In the severe cases, the entire wall of the bowel is thickened and can be demonstrated as a rather rigid tube that resists all efforts at dilatation.

Dr. McGlannan: The points brought out by Dr. Lynch seem to be on the early recognition of cancer of the colon and rectum. The cure depends on early operation, and surgeons feel sure that early recognition does not mean a limited operation, so the few hints that Dr. Lynch gave of the extent of operation will apply, no matter how early we see the patient. The most striking symptom in gastrointestinal cancer in general is the sudden onset of inactivity of the gastrointestinal canal, in a patient of cancer age. Whenever that history is obtained, we should make a careful study to discover the presence of tumor. In the case of the rectum, of course, the prominent symptom is constipation. This, coming on suddenly in any patient, demands a careful rectal and sigmoidal examination, tactile and instrumental. For the diagnosis of higher growths the X-ray is probably the safest means at our disposal at this time. We must bear in mind the possibility that contractures due to spasm may be shown by the

X-ray. These contractures may be relieved by anti-spasmotic treatment, and then a second series of pictures will prevent a needless operation. The character of the tumor in the colon above the rectum has a great deal to do with the probability of the cure of the patient. We found, in going over these cases, that there are two very distinct types of cancer of the colon: In one, the cells are cylindrical, the growth extends into the lumen of the bowel, but does not penetrate the peritoneum or metastasize until very late. Cases of this type usually come to the surgeon as the result of acute intestinal obstruction. The early symptoms have been overlooked until there is a sudden complete obstruction, and then the patient is brought in with the diagnosis of acute intestinal obstruction. We usually do an enterostomy, and at a second operation resect the cancer. The percentage of cures is good. In the other type, with cuboid cells the growth does not penetrate the lumen, but quickly metastasizes to glands, to the liver and other organs, and when the patient finally gets obstruction, as the result of infiltration and spasm, the tumor has already metastasized, and we cannot cure the patient. So we come back to the recognition of the first warnings. When the colon or rectum goes out of function, it should be examined, and if this is done by tactile examination, with the aid of the sigmoidoscope, for the lower colon, and with the X-ray, in the higher growths, excluding cases due to spasm, we shall get the cases early, with the infiltration of the wall, and before the obstruction of the lumen has occurred.

DR. J. C. HEMMETER, Baltimore: As the chairman has pointed out, as soon as you can recognize tumor of the rectum, it is too late for early diagnosis, and, commenting on what Dr. Lynch has said of the diagnosis of late rectal conditions, I would state that some years ago I investigated the cause of that late diagnosis. I simply put this question to each rectal patient: "How long after you first noticed symptoms did you consult a physician?" I found that the fault was usually with the patient. It is not a deficiency of clinical or surgical skill that is responsible for these late cases, but procrastination on the part of the patients.

I am anxious to hear from Dr. Bryant whether he was correctly quoted in regard to closure of the sigmoid.

DR. JACOB KAUFMANN, New York City: There is no doubt that in a certain series of cases, the latent period of carcinoma is so extended that the patients come under observation only when it is too late for radical removal. That is, these patients have no symptoms that induce them to consult physicians. It is also true that patients who show distinct symptoms neglect them for a time. I do not think that the stipulation of a generally conducted thorough examination will help us, unless the history of a case directs our attention to the possibility that there may be a cancerous growth. In my experience, nothing in the history of these patients is more characteristic for a possible development of a growth than the sudden abrupt onset of symptoms in a previously healthy man no matter what these symptoms may be. Dr. Lynch mentioned a case with a sudden recurrence of the carcinoma after operation. There was a sudden onset of constipation. It is the sudden onset of a symptom in a patient previously quite well that should arouse immediately the suspicion of cancer. In such cases, one should go through the routine examination, including the examination of the rectum and sigmoid by means of instruments. I have several times been enabled to make a positive diagnosis of carcinoma of the rectum and sigmoid by using the sigmoidoscope and I agree with Dr. Lynch that it is wrong to neglect to explore the rectum and sigmoid with the finger, or the proctoscope, and be satisfied with the result of X-ray examination, which may mislead one.

Dr. Frederic Kammerer said he did not entirely agree with Dr. Hemmeter. Ever since he had been associated, for a number of years, with Professor Kraske, the originator of the method of excision of the rectum known as the Kraske operation, the speaker had been interested in the subject of rectal carcinoma, but even now he not infrequently saw cases of far advanced cancer, as he did thirty-five years ago, in which no diagnosis had been made, and not even a rectal examination. Where hemorrhages occur in cases of rectal cancer the disease generally is pretty far advanced and easily recognizable by the finger introduced into the rectum. It is regrettable that rectal exploration is still so often omitted in these cases, which are treated as hemorrhoids.

Constipation, of course, is a valuable symptom, but it should be borne in mind, in this connection, that small constricting carcinomata, in the large intestine, of slow growth, can exist for a long time and narrow the lumen of the bowel appreciably without causing marked symptoms of constipation. This is due to a hypertrophy of the muscular coats of the intestines above the stricture. Very often symptoms of acute obstruction first direct attention to the presence of a growth.

The speaker said he always used the Kraske incision in cancer of the rectum, but had given up the typical resection. In the discussion of a paper, read by Dr. Tuttle many years ago, in which cases of resection of the rectum were demonstrated, he stated that strictures of moderate degree always occur after resections. In many of his own cases he had also noticed that recurrences are apt to occur at the point of resection. He did not say that the narrowing of the gut is essentially the cause of these recurrences, rather, in doing a resection, we do not perform as thorough an operation as when we extirpate the whole lower segment of the large intestine.

The speaker did not agree with Dr. Lynch regarding the artificial anus. He thought the patient was better off with an artificial anus in the left inguinal region. He had accepted this procedure in all his operations during the last fifteen years. In former years he had given rather an extensive trial to the various methods, destined to promote continence of feces, when the anus with its natural sphincters has been removed and the end of the bowel placed somewhere near its original site. In such cases results were not satisfactory, and he had come to the conclusion that an artificial anus in the inguinal region is always under better control of the patient. The latter can take an enema once a day, or every other day, and, with proper regulation of diet, the artificial anus gives little trouble, especially after some time has elapsed.

DEVELOPMENTAL RECONSTRUCTION OF THE COLON: ANIMAL RESEARCHES AND CLINICAL REPORT OF TWENTY-NINE HUMAN CASES.*

By John William Draper, M. D., F. A. C. S.

Comparative anatomy sheds an interesting and instructive light upon the development of the human colon.** Huntington,† discussing primitive types and in particular the terminal straight segment of the canal which is so much shorter in many vertebrates than in man, says: "The primitive condition of the intestinal canal may be observed in some members of man's own class, the mammalia, as in certain edentates. Notable among these is the little anteater of Brazil. The termination of the duodenum and the bend in the colon mark the two points at which in the primitive scheme the umbilical loop begins and terminates. These two points we can describe as the duodeno-colic neck or isthmus. In the snapping turtle the same condition prevails—namely, the duodenum and the colon approach each other very closely at the isthmus and between these points the convolutions of the intestine extend in a wide circle. We will find this approximation of duodenum and colon a feature which persists throughout all the later developmental stages of the higher vertebrates and has an important bearing on the final arrangement of the intestinal canal in the human adult."

Thus not only throughout the fish, the turtles and the frogs, but actually in man's own class, we find types in which the adult corresponds in colonic position and limitation of growth to the primitive form.

Anatomy and embryolgy are not alone in giving important information as to the relative value to the human organism of the right and left sides of the colon. In a paper read before the section on Pathology and Physiology of the American Medical Association, 1914,* I have called attention to the relative inactivity of the right or oral colon in the dog as regards the excretion by it of drugs and toxines compared with the activity of the left or aboral portion of the organ. Briefly, it has been noted by us in company with many other, that even in an animal like the dog in which there is normally no ascending colon and a rudimentary cecum, there is a vast difference between the excretory activity of the ceco-colonic region and the caudad portion of the organ. In pilocarpine or diphtheria poisoning, or in the toxemia of duodenal obstruction, the oral portion of the colonic mucosa remains normal in color while the aboral assumes a brilliant scarlet.

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*Read before the American Gastro-Enterological Association, Atlantic City, May, 1917.

**The Anatomy of the Human Peritoneum and Abdominal Cavity, p. 56.

†From the Laboratory of Surgical Research, New York University and from the Clinic of Surgical Gastroenterology, New York Polyclinic School and Hospital.

A third avenue for the approach of this interesting problem of colonic variability is found in clinical study at the operation table on adult human beings. It has long been known to biologists that variations in form attest to recency of origin and instability in function. What thoughtful surgeon is there among us who has not been repeatedly struck with the amazing variations in form on the right side of the colon as contrasted with their absence on the left. The occasional exception to this occurs only in pelvic colon. Moreover, not as a rule having had opportunity for the study either of embryology or of comparative anatomy he has been at a loss to comprehend or to make any reasonable interpretation of these variations.

It was only after many years' consideration of these abstract phenomena that my colleague, Jerome Lynch, and I became interested in the possible meaning attached thereto and as to whether any utilitarian application thereof could be made in improving the therapeusis of human disease. Especially after reading Madison Grant's notable book,** however, we are more than ever convinced that such utilitarianism, be it in pedagogy, in medicine or in other walks of life, is one of the poorest, if not the very worst, characteristic handed down to us from the Nordic race and of very secondary value as compared with search for the abstract truth. We hold with increasing tenacity that no lasting progress can be made in medicine except by approaching

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*Draper, J. W.: Studies in Intestinal Obstruction. *Journal A. M. A.*, September 26th, 1914, page 1079.

**The Passing of the Great Race.

its great problems solely from the standpoint of pure science. As Le Pere Felix says: "*Nos progres sont pleins de la sueur des siecles passes.*"* It is in every case an idealist; a Pasteur or a Hunter who has furnished the framework upon which materialism has built. William Salter,** speaking of the error of the general belief by which the great idealist Nietzsche, who in reality proposed a far-reaching superhuman faith, is wrongly charged with advocating this great war, says: "Can the Englishman become more than English, the German more than German; yes, can our swirling democratic masses, mad for the lesser goods of life, comfort, wealth, luxury, ease—can they rise above themselves? Can they, can we all, care supremely for this *species* of ours and some culmination of its possibilities?" As compared with abstract truth, even therapeutic improvements may rightly be classified as among the lesser goods of life. Those who make utilitarian application of abstract medical truths are not in the same class with those who seek them.

In a recent unpublished address before the New York Academy of Medicine the English Physiologist, Haldane, said that the modern mechanical methods in physiology had about reached the limit of productivity and that a return would shortly be made to some of the methods previously in vogue, and long since discarded by the mechanistic school, notably the theory of vitalism and its allied branches. We are at the parting of the ways.

From our laboratory and clinical studies we are convinced that our provisional hypothesis regarding the ceco colon, its relative unimportance and frequent danger to the human economy, based as it was upon biologic premises, is correct. Our clinical data and our experimental studies agree with these premises. The two support each other. Bryant's studies† resulting in a classification of human beings and diseases into herbivorous neutral and carnivorous types, as well as the well-known work upon right colonic resection and exclusion of Bloodgood, Mayo, Ochsner, Satterlee and others, compel the general attention of the profession to the ceco colon. The far-reaching variations in the syndrome of human symptoms, varying in all grades from the neuro-mental to the arthritic, and which in properly chosen cases are cured or improved by right-sided resection show the problem to be one of the most fundamental in all medicine. Obviously it can be solved only by the combined efforts of both clinical and biological workers; the bio-clinician having naturally the greatest opportunity for success.

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*Conferences de Notre-Dame de Paris.

***International Journal of Ethics*, Vol. 27, April, 1917, page 378.

†Bryant, John: The Carnivorous and Herbivorous Types in Man. *Boston Medical and Surgical Journal*, September 9th, 1915.

Experimental Studies.—These were made on dogs and were of necessity limited in scope owing to the embarrassing fact that the ascending colon is normally lacking in the dog, and that like the anteater its duodenum and colonic bend are closely approximated. Thus the colon is exceedingly short and affords but limted chance for operative procedure. We have been, therefore, confined to a technical consideration of the effect of the modified developmental reconstruction which is possible in the dog upon the motility of the stomach as shown by X-ray and upon the relative merits of the lateral, the end to side, and end to end forms of anastomosis, as observed by the same agent. The accompanying table is self-explanatory and supports in general what has been the usual holding in human surgery; that whereas the end to end technique gave the most ideal result, its high mortality made necessary a choice of either the lateral or end to side implantation.

No surgical research on animals can possibly be made without the conclusion that the persistence in man of the right colonic segment which has come to us apparently through the dominance of its experimental lengthening by the herbivora is ill adapted to our needs. Just what relation the upright position may have to the frequency of localized right-sided constipation, we are not prepared to say, because so little is known of the relations between gravity and intra-abdominal pressure. Constipation unquestionably is merely a part of the protective bodily mechanism. Having learned largely through Barber's researches* that gravity has little or nothing to do with the emptying of the stomach after gastro-enterostomy, stoma position and neuro-muscular gastric condition being the true determining factors, we are very loath to place much credence in the popular but ill supported theory that the human cecum often does not empty because of gravity. We are, of course, ready to concede that this factor has its

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*Barber, W. H.: Segmental Resection for Gastric Ulcer. *Annals of Surgery*, November, 1916.

DEVELOPMENTAL RECONSTRUCTION.

Before Operation.			After Operation.			
A	Elapse of Time	Remarks	A	Remarks		
End to End Anastomosis—3 Cases	Between Meal and X-Ray A	Stomach Emptying	Elapse of Time	Stomach Emptying	Anastomosis Emptying	No. of Days Post Op.
No. 1. Dog No. 170	2 hours 50 minutes	Slight Residue	3 hours 20 minutes	Normal	No Delay	7
No. 2. Dog No. 174	3 hours 40 minutes	Slight Residue	3 hours 25 minutes	Delay	No Delay	13
No. 3. Dog No. 191	2 hours 50 minutes	Slight Residue	3 hours 20 minutes	Delay	No Delay	14
B						
Side to Side Anastomosis—3 Cases	B					
No. 1. Dog No. 175	3 hours 40 minutes	Empty	3 hours 20 minutes	Marked Delay	Slight Delay	10
No. 2. Dog No. 192	2 hours 50 minutes	Slight Residue	3 hours 40 minutes	Marked Delay	No Delay	12
No. 3. Dog No. 203	3 hours 00 minutes	Empty	3 hours 00 minutes	Marked Delay	No Delay	12
C						
End to Side Anastomosis—3 Cases	C					
No. 1. Dog No. 186	3 hours 5 minutes	Residue	3 hours 15 minutes	Moderate Delay	No Delay	13
No. 2. Dog No. 198	3 hours 10 minutes	Residue	3 hours 10 minutes	Slight Delay	No Delay	10
No. 3. Dog No. 206	3 hours 5 minutes	Residue	3 hours 5 minutes	No Delay	No Delay	6

place in the causative syndrome, but realizing the markedly close analogy both as to physiology and as to morphology which exists between the stomach and the cecum, and to which we have repeatedly referred in previous articles,* we feel quite certain that the gravity hypothesis is at best inadequate. Our hesitancy is further increased by the findings of our colleague, McFarland, whose pathological studies** of the ceco-colons removed by us show the tissue to present certain atypical characteristics notable among these being the frequent presence of pigment. This we regard as a highly important contribution.

This affords additional basis for the assumption that an inherent biological instability of the ceco-colon as developed in man is more responsible for the symptoms which unquestionably arise from it, than the effect upon its emptying by gravity. There is undeniable decadence in function in all organs developing in the higher types in late fetal or early extra-uterine life. We must all remember that colonically speaking we are dogs at birth, the colon reaching only to the liver; thus the left colon, excepting only the pelvic portion, is of extreme age, of constant and important function, of fixed morphology as compared with the right which has not these important attributes. May it not be more logical to consider the relative importance of the right and left sides from the broad standpoint of phylogany rather than from the narrow viewpoint of descriptive anatomy, and to base our therapeusis upon known biological laws rather than upon unproved and very questionable clinical hypotheses. This must be even more acceptable if the biological laws prove to be supported by our clinical therapeusis in individual human beings. Thus the ceco-colon may be considered the decadent wisdom tooth of the alimentary canal and be treated as such.

Clinical Report.—In twenty-nine human cases of developmental reconstruction of the colon five have died, a mortality rate of 17 per cent. An analysis of this mortality is proper at this point because its degree would be deterrent and properly so in the employment by others of this operation. Four deaths occurred in the first eight cases, one death in the last twenty. An analysis

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*Lynch-Draper: The Protective or Esoteric Symptoms of the Alimentary Canal. *Virginia Semi-Monthly*, March 24, 1916.

**McFarland, W. L.: Pigmentation of the Hind Gut. *Journal A. M. A.*, December 8, 1917.

shows two important facts. First, that we did not understand how to chose the cases. Second, that our technique has improved. The cause and time of death in the five cases is as follows: In two, both of them epileptic, shock in less than 24 hours after operation. In one, hemorrhage 6 hours after operation. In one septicemia, four weeks after operation. Autopsy showed an old peri-nephritic abscess, unrecognized at time of operation, as probable cause of death. One in a debilitated derelict, five days after operation; peritonitis. We have eliminated much of the postoperative shock by careful avoidance of all traumatism to the nerve centers near the duodenum. The postoperative hemorrhage death was due to the nurses' failure to report patient's conditon until too late. The peri-nephritic abscess case in which death occurred four weeks after operation and in which all bowel functions had been completely restored to normal, should not be charged against the series except in an indirect manner. Reclassifynig the cause of death, therefore, we find that peritonitis, due directly to leakage from the anastomosis caused one death, or 3.4 per cent. Shock in two epileptics, or 6.8 per cent.; septicemia from long-standing infection, or 3.4 per cent.; accidental hemorrhage, one death, or 3.4 per cent. This analysis is in no way an attempt to minimize the danger and gravity of this operation, which we feel sure should not be undertaken except after the most exhaustive studies; until competent and long-continued medical care has proved unavailing and then only by an experienced operator.

Effects of developmental reconstruction upon the patient's symptoms. In order to study these it is necessary to make an attempt at symptom grouping. For our purposes a classification of the patients into two genral groups has served the purpose. The first, and by far the larger group, may be termed the "Neuromental"; the second, much smaller, the "Arthritic." This is an arbitrary setting which perhaps should be discarded and can no doubt be improved upon.

Something should be said regarding the methods employed to group the cases and to reach a decision as to operation. We consider it imperative that each patient should be studied by X-ray, and that the operation is indicated in exceedingly few cases that do not show ceco-colonic delay of 100 hours or more. The symptoms certainly are not due to constipation, which we regard definitely as a protective symptom, but rather to the diseased state of the ceco-colon which permits passage of toxines arising either from bacteria or from the gut wall itself. This latter occurs in duodenal obstruction. Why not in colonic? Satterlee's studies* of the chemical blood picture in colonic toxemics (human) are of the utmost value and interest tending, as the accompanying tables show, to indicate that a toxic element arises under conditions of partial or complete obstruction of the terminal gut similar in effect to that of duodenal obstruction, already published by me.

RESULTS OF EXAMINATION OF SIXTEEN CASES OF CHRONIC INTESTINAL TOXEMIA IN WHICH BLOOD WAS EXAMINED CHEMICALLY.

Non-protein nitrogen	Over normal (45 mg. per 100 c.c.)	14 cases
	Under normal (30 mg. per 100 c.c.)	6 cases
Urea nitrogen	Over normal (25 mg.)	4 cases
	Under normal (15 mg.)	6 cases
	Normal	10 cases
Uric acid nitrogen	Over normal (3.5 mg.)	4 cases (highest 10.49 mg.)
	Under normal (1 mg.)	8 cases
	Normal with subsequent rise	1 case
	Under normal with subsequent rise	1 case (sudden gout)
Creatinine	Over normal (0.5 mg.)	12 cases
	Under normal (0.1) sudden rise	1 case
	Normal (0.1-0.5 mg.)	7 cases
Blood sugar	Over normal (120 mg.)	6 cases
	Under normal (50 mg.)	0 cases
	Normal (50-120 mg.)	14 cases

We also regard as imperative not only the removal of all possible areas of infection due to local foci, tonsils, teeth, cervices, etc., but even more particularly the repair of any and all local lesions such as hernia, hemorrhoids and the like which may have important bearing through reflex inhibition of the right side of the colon. Needless to say every case should be put through a complete laboratory study, including the Wassermann reaction. In one of our cases this unfortunately was not done, dependence being placed upon a 100-hour right-sided X-ray delay. Developmental reconstruction reduced the barium oro-anal time to 36 hours due to the fact that the operation had enabled us to remove an extensive area of partial obstruction at the hepatic flexure; Lynch's "Elbow Deformity." The patient's symptoms of pain referred to the rectum, however, persisted until we all, as he said, thought it was "in his head." An "ex post facto" Wassermann, however, was three plus.

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*Personal communication.

Our earlier cases now date back nearly three years. The greater number are over one year and a half, the most recent is three months. It is, therefore, probably justifiable to present some utilitarian deductions based upon subjective and objective data. Careful and conservative study of the twenty-four living members of this interesting series shows at the very least a 75 per cent. improvement in efficiency and subjective symptoms. Incidentally the more we study these cases the greater respect we have for the subjective testimony. The extraordinary accounts by these highly neurotic and often unbalanced patients have in the past been looked upon as worthless, but we have come to regard them with great respect, having often found at operation objective corroboration. Truly, as Wm. Mayo has well said, in rejecting such testimony as a worthless fable, we ourselves are indeed the fool rather than the patient.

Technique.—We have resected the terminal 10 cm. of the ileum and the ceco-colon in block to the neighborhood of the right colic artery. This corresponds to a point upon the transverse colon near to the right margin of the omentum. Since in practically all of our cases there has been a common mesentery of the cecum and ileum so that the entire mass, particularly after careful separation of the mesenteric leaves, could easily be lifted above the belly wall so that both the resection and the anastomosis was done extraabdominally, we have been able to avoid shock by keeping away from the duodenal centers. Keith's Center at the terminal ileum, while doubtless in close and important neuromuscular relationship with the duodenal centers as proved by the interesting and conclusive experimental researches of Barber, may apparently be removed without adding surgical shock. This does not mean, however, that we consider ourselves at all informed as to the final postoperative result of its loss to the economy.

Case Histories.

Neuro-mental Group: Mrs. R. B. E. This intelligent woman is from the professional ranks and has had the best medical care. She was operated on two and a half years ago. Age 28. Two healthy children 8 and 10 years old. Chief complaint: Chronic constipation associated with extreme mental depression—suicidal; in the State Insane Asylum, Waverly, Mass., 6 years ago for six months. Husband was told by the alienists that she never could be cured; a hopeless mental case. Now reports herself 80 to 90% improved and able to care for her household and social duties; bowels regular; no cathartics. When presented recently before the Medical Progress Club of New York she replied to an inquiry as to what her chief trouble had been before the operation "trying for 15 years to keep from losing my mind."

Miss A. J. L'E. This woman is a descendant of an old Belgian family and knows her mind. Before operation she had been in the hands of a trained nurse under the most competent medical supervision that money could obtain. She had traveled with the nurse from the Gulf of Mexico to Laborador in the vain search for health. She was regarded by her family and attendants as deranged. Without provocation she flew into the most violent fits of uncontrollable rage. It was impossible for her to associate with anyone. She complained of the most frightful headaches, hemicranial in type. Her bowels were irregular, constipation alternating with diarrhoea. Two years and eight months ago developmental reconstruction was done. Her ceco-colon was grossly and microscopically atypical being filled with pigment, and the mucosa being nearly destroyed. That this woman's symptoms were directly attributable to the absorption of unknown products from this degenerated and sac-like bowel segment is undeniable, for not only has she returned to her arduous work of teaching, but her headaches are 95% diminished, her bowels are regular and she is able to associate with her fellow beings.

Mrs. M. E. R. This woman, 44 years of age, was for years a chronic sufferer with hemicranial headache; in bed for five days with each attack. In 1909 she had 67 headaches; in 1910, fifty-nine; in 1911, sixty-five; in 1912, only thirty-five (this she attributed to rigid abstinence from meat); in 1913, forty-nine; in 1914, fifty; in 1915, sixty-five (the diet had been rigidly maintained but the headaches had now reached their former maximum). Developmental reconstruction 6 months ago. Constipation cured; one headache only which patient attributed to her over-indulgence socially. Of particular interest from a bio-chemical standpoint is the fact that she has added both meat and eggs to her diet. For the past 6 years she had not been able to eat an egg without being certain of having a headache.

Arthritic Group: We have had but one case presenting uncomplicated multiple arthritis. We offer no explanation for this curious evidence of selective action of the toxines. It has happened that this case was evidently well chosen for she has been rendered 80% efficient by developmental reconstruction of the colon. While the arthritic was marked in the lower extremities the patient could still walk by a sort of creeping gait. She had not been able to feed herself except occasionally with a spoon for over 2 years. She had not been able to turn over in bed for 2 years; she had not been able to write for 3 years. After every possible source of infection had been sought for elsewhere it was decided that the toxines were being distributed from her colon where she presented 100 hr. right-sided delay. Sevnteen months ago developmental reconstruction was done. In 36 hours the patient affirmed that every vestige of pain had left her body and that it was the first time in 3 years that she had not suffered pain day and night. More remarkable to us was the change about the small joints. How the scarlet color could be blanched as it was in 36 hours still remains a mystery. We are aware that Bottomley and others have not had as gratifying results as we are able to report in this case. Whatever the future of this individual may be as to relapse and recurrence, the study of her postoperative condition is of the utmost value in proving that in her case at least the toxines did come from a diseased ceco-colon.

Indications for Developmental Reconstruction.

1. Segmental infection of the ceco-colon. Like any other infected hollow organ it is a constant source of danger.

2. In Lynch's elbow deformity of the mid-ascending colon when the peritoneum cannot be repaired after reduction.

3. In exaggerated non fusion of mesentery, allowing such freedom of motion as occasionally to result in volvulus. This happened in one of our series.

Conclusions.

1. Developmental reconstruction of the colon is an operation which finds justification in heredity, in well known laws of biology and in clinical study.

2. It is a procedure fraught with danger and one which never should be undertaken until, not alone exhaustive studies have been made but until modern and well directed medical therapeusis has failed.

3. Ileo-sigmoidostomy is unphysiologic and has therefore failed.

4. The subjective symptoms for which the operation is indicated are usually neuro-mental; the objective symptom is

ceco-colonic degeneration. The degree of improvement in our series is amply sufficient to justify the operation in selected cases.

5. Developmental reconstruction has superseded in our clinic all the earlier forms of operative procedure such as ileo-colostomy, ceo-sigmoidoscopy, etc., because we have found it to be no more dangerous operatively and to give better results because it removes the biologically decadent and diseased organ and restores the individual to the carnivorous type of colon undoubtedly best suited to man.

DISCUSSIONS.

DR. *Bryant*: A number of persons have an idea, apparently, that remarks I have made on the subject of type in the human show that these investigations are in themselves an end object. They are not. They are a side issue, in an attempt to find out what is the matter with the chronic intestinal invalid of the thin type. This problem boils down practically to a developmental proposition; that is, the so-called thin type of people have for example an arrangement of the intestine that is developmentally delayed. If one goes back to fetal life, one finds that the sigmoid loop in the early fetus is always practically one-half of the large intestine. That excess gradually diminishes; and the people in whom this loop goes to the diaphragm, as we saw in the picture shown yesterday, are, so far as I can see, subjects of faulty or arrested development and differentiation.

Dr. Draper spoke of one interesting developmental question, and that is the early points of attachment of the colon: first, the duodenal flexure; and second, the splenic flexure. I saw, among a considerable series of autopsies examined two or three years ago, a woman of sixty-seven or sixty-eight years, in whom there was absolutely no attachment of the intestines below the duodenal junction. She was apparently in good condition. Everything was loose, but there were no adhesions. That was apparently a very late specimen of the developmental proposition, without even the splenic flexure attached.

Going now to adult life, we find that the fact that the splenic flexure is firmly attached is quite important; because associated with a long sigmoid loop and a high splenic flexure, one gets very frequently a low hepatic flexure and cecum.

In regard to the question of what is to be done, I think that if one after conscientious medical treatment is forced to surgery, it is pretty certain that right-sided colectomy is best. Dr. Draper spoke of the "wisdom tooth of the bowel," and I should like to limit that term to the caecum and hepatic flexure. A very large percentage of visible adhesions come within a circle in the right hypochondrium, which includes the first portion of the transverse colon. The appendix adhesions are much less in number; and if one removes the right half of the colon, one removes a large percentage of demonstrable adhesions, whether developmental or acquired.

I believe that most of them are developmental.

In regard to treatment, one may have all sorts of angulations, and yet have no apparent trouble. I do not see any reason for bothering about these findings if there is no trouble caused by them, but I believe that they do *predispose* to trouble, and that, in the presence of such conditions as fatigue, the person with angulations and adhesions is much more liable to go under than is the person without them.

There is one thing that has not been mentioned, perhaps, as much as it might be, and that is the condition of the ileocecal valve. There should be a break in continuity between the terminal ileum and the colon. If this exists, it is much less important what you do with the colon and what you put in it. Delay is of much less significance than is an absence of this barrier. That is one thing that should be considered in deciding this question of right-sided colectomy.

I have seen a number of these operations performed in people in whom it has taken the following year or two to develop the condition, but there is then frequently a dilatation of the terminal ileum, that acts as a sink, so that you may have infection originating in this neighborhood. I have also seen a great deal of "nerve" trouble in these patients clear up under appropriate dieting.

Dr. Bryant: I agree with Dr. Lynch. It was improper for me to use the word "valve." It is absolutely correct to call it a sphincter. It was a slip of the tongue on my part.

Dr. J. W. Draper gave a lantern demonstration which he discussed as follows: He had been much interested in conducting experimental research on dogs with a view to determining the effect of the three kinds of anastomosis, which may be employed after developmental reconstruction of the colon (right-sided resection), namely, lateral, end to end and end to side. The two obvious considerations in this matter were the mortality rate and the efficiency as determined partly by gastric residue and partly by local residue at the anastomotic site. Briefly, while the end to end was the most brilliant of the three, the lateral was by far safer, the end to side occupying a mid position. So far as the residue occurring about a lateral anastomosis, the speaker said that although it was often large, he had been unable to prove that it was attended with serious clinical consequences.

He then showed a picture which presented the origin of cecal diverticulae and urged the importance of regarding these as embryonic remnants and therefore in a large measure physiological. In certain of the fish, notably in the ganoids and teleosts, and particularly in types without a spiral valve which is a device for increasing the surface of the gut, often as many as 150 diverticulae occurred normally near the pylorus. These were of evident value and there could be no doubt that in human recessional forms diverticulae might not only be harmless but useful. Embryology was a good basis for the interpretation and explanation of what we are apt to consider abnormal.

He then showed a series of slides exhibiting ileo-sphincteric leakage after enema. He and Dr. Lynch had been much interested in giving the patients a hypodermic of epinephrin. It was found that in a large per cent. of cases this secretion closed the valve tight, and it was not improbable that this would be a means of clinically discriminating between ileo-sphincteric leaks, due to actual mechanical lesions and the much more numerous class due to internal secretory deficiency. At any event he did not consider leakage, unless under exceptional conditions, a proper index for laparotomy; that it should be looked upon as in some measure protective rather than otherwise, and that the above experiments led to this view.

He then showed a series of slides illustrating the results after the operation of developmental reconstruction of the colon. He and his associate, J. M. Lynch, had performed this operation on twenty-nine human derelects. He spoke of the desirability of prolonged postoperative study in these cases. A number had already been watched for three years. Some of course had been failures, but in a great majority sufficient improvement had been noted to warrant the belief that many of the ordinary symptoms, both nervous and mental, as well as much of the physical inefficiency among us could be traced directly to the colon. Howard Barber's recent researches proving the close neuromuscular relationship, which exists between the beginning and the end of the alimentary canal have gone far to prove the fallacy of considering localized areas of the canal instead of the organ as a whole. Thus although there might well be absorption from the ceco colon owing to impairment of its epithelium, as was indicated by the frequent presence of pigment studied and reported by McFarland in the specimens removed by the speaker, it was altogether probable that the interferences with the calibre of the duodenum coming reflexly from a diseased ileocecal region might well cause in addition absorption of equally poisonous, non bacterial biochemical toxines known to originate from the duodenal epithelium under obstructive conditions.

Another series was devoted to the postoperative study of some of the cases of developmental reconstruction. The first showed failure of rotation and coincident tuberculosis which involved the terminal ileum and supported the speaker's contention that this portion of the small gut having a common embryologic origin with the colon should be expected to share in its lesions as well as under certain conditions which he had proven experimentally to assume its function vicariously. One patient had been operated on deliberately for hemicrania. She had kept an accurate record for seven years of the number of headaches that she had. At first they averaged 60 a year. Her doctor deprived her of proteins and the headaches immediately dropped to 30 a year. After that they gradually rose to 40 a year for some time until in 1916 she had 60. There was no doubt that hers was the proper case for developmental reconstruction since a most careful physical and laboratory study failed to reveal any other lesion whatsoever. This patient was operated on a year ago and has recently reported herself as headache free, a 100% cure.

Finally a colored slide was shown to illustrate the difference in reaction of the left side of the colon in the dog to pylocarpin poisoning and to duodenal obstruction. These studies prove that many poisons including that of intestinal obstruction, were thrown out by the left colon, and that its removal should therefore never be considered except for megacolon or for cancer.

THORACIC STOMACH—GASTRO-DIAPHRAGMATIC HERNIA.

With Report of a Case of Probable Congenital Origin.

By J. Harry Ullrich, Ph. G., M. D., Associate Gastro-Enterologist, University of Maryland.

Henry J. Walton, M. D., Roentgenologist, University Hospital,

BALTIMORE, MARYLAND.

This case is of unusual interest, because the first thought of a physician who sees, hears or reads of a case with the symptomatography of anorexia, nausea, expulsive vomiting, gastralgia, rapid and progressive loss of weight, and the patient aged 26 years, is not of such deformities as diaphragmatic hernia, eventration, volvulus or diverticulae of the esophagus.

Diaphragmatic Hernia.—May be congenital, traumatic or acquired, of which the former is reported to be the most frequent form.

Case Report: The patient, Mrs. S., a resident of Baltimore, is married, aged 25 years, at the time she first came under my observation, May 2nd, 1916. On account of my absence of 7 months from the city, incident to Medico Military Service on the Mexican border, from June 19th to January 6th, 1917, I did not see her again until February, 1917, during which time she had lost about 40 pounds in body weight. Gastric analysis revealed acid secretion to be slightly below normal. One skiograph, taken in May, 1916, which will be presented by Dr. Walton, shows the stomach abnormally high, apparently transposed and not clearly defined.

Chief Complaint: Inability to retain food, liquid or solid, without pain, or the sensation of wanting to vomit; that regurgitation was so expulsive, that she was compelled to hold her hand over her mouth, and rush to a place for gastro-oral evacuation to avoid accidents and embarrassment.

Family History: Father died aged 71 years, cause neuritis; mother living and well, aged 83 years, but partially blind; nine brothers, all living and well; two sisters, one living and well; the other died at the age of 29 years, from cancer of the breast.

Past History: The mother of the patient advises that she was a very delicate child at birth, and continued as such during her infantile life; vomited whenever food was taken. The patient reports that, as far back as she can remember, that she had frequent attacks of vomiting, which usually took place during the intake of food or followed immediately afterward, and that whenever food was eaten and followed by sensations of distress, such as nausea, oppression or pain, without involuntarily vomiting, that it was and is her habit to superinduce vomiting, by putting her finger down her throat, which easily caused vomiting, and relieved the distress. Had a severe attack of whooping cough when an infant; measles at 5 years, with a subsequent attack one year later, which was very severe; she had rheumatism at 7 years, involving all joints from the hips down, duration about two weeks; slight attack of gout in 1910, duration about two weeks; married in 1910, but has never been pregnant; constipated all her life, which is undoubtedly due, at present, to deficient food ingestion, both liquids and solids.

History of Present Illness: Up until May, 1916, she enjoyed eating, and had a fairly good appetite for food, solids and liquids; had always looked lightly upon her condition, as there had been an absence of severe pain, vomited almost daily with ease and relief, whenever stomach discomfort was experienced. Anorexia developed, which was noticed and commented on by other members of the family, and in an effort to please her aged mother she frequently forced herself to eat, and suffered accordingly. Solid foods caused distress and pain, particularly meat, as did bread and potatoes, till vomited; milk would be vomited as a coagulum (thus evidencing gastric action), liquids were and are better borne if taken in small amounts and at frequent intervals. Since January, 1917, has had, after retiring, several severe attacks of gastralgia, and vomiting; expelling large amounts of sour and offensive fluid. Physical signs, such as dysphagia, dyspnea; unilateral prominence, comparative motionless or restricted expansion of the lower portion of the chest are all negative in this case.

Physical Examination: Aged 26 years, height 60 inches; weight, May, 1916, 155 pounds; January, 1917, 117 pounds; April 1, 1917, 111 pounds. Patient appears weary, fairly well nourished, evidences a slight nervous tremor of the hands. The skin is pale and sallow, but betrays no evidence of a cyanosis about the ears or finger tips; mucus membranes of the eyes and lips are apparently normal and well injected. *Tongue,* moderately hypertrophied and flabby, with impressions of the teeth on each side, heavily coated and of a dead color. Teeth are normal in conformation and none-defective, but breath is very offensive. *The eyes* are rather prominent, inter-ocular tension slightly increased; sclera normal in color; marked hyperopia but corrected by and with glasses.

The Thoracic Examination: As to heart and lungs negative. There is, however, an area just above, and to the right of the xiphoid which on an empty stomach is hyperresonant on percussion, the note changing to flatness after drinking a glass or two of water; gurgling and splashing heard on asculation: posteriorly the same changes were noticed but less marked.

Deglutition sounds—heard anteriorly in 4 seconds.

The Blood Pressure Is: Systolic, 116; diastolic, 76.

The Abdomen: In all its regions is soft and pliable, except to the left and upward for about three inches above the umbilicus, which is slightly tense and sensitive to palpation. On percussion, a condition of tympathy is revealed from *two inches above the xiphoid to the hypogastric region,* and is interesting in that there is not any change of tone in the percussion note (hyperresonance) between these two points; doubtlessly due to gas or air inflation of the hernated stomach in the thorax, and of the intestines below.

Lymphatic glans negative to palpation.

Gastric Analysis: When the stomach tube was passed on three or four different occasions following an Ewald test meal, same could not be aspirated after the expiration of 45 minutes. It was noticed that after about seventeen inches of the tube had passed that a slight resistance was met, but the tube, upon slight pressure could be advanced the normal distance without causing pain. Upon one occasion the tube was gently advanced about three inches beyond the normal length, with the result that 50 c.c. of gastric contents, fairly well chymified, and of normal color, odor and appearance was withdrawn, acid in reaction to dimethylamidoazobenzol (which contraindicates a condition of food or contents from sacculation or diverticulum of the esophagus). The patient reported the next day that she felt that the tube had reduced a sack and that she never felt better. Specimens of vomitus taken on several occasions all showed gastric action, as did all Ewald test meals, that were aspirated.

Chemical Analysis: Free HCl 16, total acidity 38. Tests for blood negative.

Microscopically: No evidences of fermentation (yeasts) or malignancy (Oppler Boas bacilli) were seen.

Blood Examination: Hemoglobin, 85; leukocytes, 9,400; red corpuscles, 4,200,000; polymorphonuclears, 49%; small lymphocytes, 30%; large multinuclears, 9%; large lymphocytes, 10%; transitionals, 2%.

The Stool: Light in color (patient on a meat free diet) negative for occult blood by the benzidin test.

The Urine: (24 hour specimen) 12 ounces, shows a large amount of indican, a specific gravity of 1030, acid in reaction, negative to albumin and sugar.

Prognosis: Exceedingly grave, from any one of the following conditions: (a) Strangulation, (b) Acute Dilatation, (c) Distension with pressure against the heart, (d) Pregnancy.

Treatment: Surgical.

DISCUSSIONS.

DR. J. C. HEMMETER, Baltimore, Md.: This very interesting case of Major Ullrich's has been studied with admirable punctiliousness. The patient has been under Dr. Ullrich's observation for several years, and the radiograms are more than radiograms; they are radiographic experiments, because he has seen the alternate filling, on tilting of the patient, and evacuation of one side of the stomach into the other half, and occurring above the diaphragm. Dr. Walton, who demonstrated the radiograms, did not suggest the point that he himself fluoroscopically observed the stomach, on one occasion, partially above and partially below the diaphragm.

Then I wish to inquire of the surgeons present concerning a certain point. I gained the impression that it was a dangerous thing to sew the stomach up near the diaphragm or close the enlarged diaphragmatic foramen, through having heard that Dr. A. D. Bevan, in operating in this way upon the stomach, does not sew the rent up, but simply fixes the stomach against the abdominal wall. I should like to be informed in regard to this.

Then the discovery of pepsin and hydrochloric acid in the thoracic stomach, when only a very short length of tube had been introduced, is interesting. This constant presence of HCl is an argument for the fact that Dr. Ullrich was dealing with a thoracic stomach and not with a diverticulum of the esophagus. This conclusion is based upon years of observation of this patient. The lady, as a child, had a very severe case of whooping-cough, which may have been a factor in the dynamics of forcing the stomach through the diaphragm. With the help of the anatomist I went to the dissecting-room and examined twenty cadavers in the dissecting room, and in every one it was possible to force the index finger into the triangular space in front of the esophagus where it passed through the diaphragm.

Dr. F. B. Baetjer, Baltimore: I should like to ask one question, and that is why Dr. Ullrich did not speak of this as esophageal dilatation with patent cardiac opening? One would be more inclined to think, from the plates that this condition was present. In all the plates, you get a good size stomach on that side. It is true that in esophageal dilatation the bismuth goes through so slowly that it does not fill up. Dr. Ullrich did not bring out this point. I should like to know whether he does not consider it esophageal dilatation of the stomach.

Dr. Ullrich, closing: The point of Dr. Baetjer is well taken, because we had that seriously under consideration for a long time during the course of our study. The mere fact that we have the colon drawn high up and gas showing proves that the bag we saw on the left, which could have been taken as stomach, was colon, which would be a natural consequence of hernia through the diaphragm, pulling the colon up with the stomach. The clinical findings upset esophageal conditions in that all food that we have been able to aspirate or catch on vomiting has been acid in reaction. (?) The patent openings began in the esophageal opening, but there was no trouble in putting the tube in any position I chose before the fluoroscope, either on the right or left side. We filled the Politzer bag with bismuth, and tried to force bismuth into the pyloric end. The patient immediately wanted to vomit, and pulled the tube out of the mouth, showing that the pressure below the diaphragm caused this pain and desire for relief. The introduction of the stomach tube on this occasion brought decided relief for several days. We had X-rayed, and found the pyloric end of the stomach below the diaphragm more than before. We found what we interpreted as a slight slipping of the various sizes (?) of the bags on each side that we were viewing through the fluoroscope.

DR. FRANK SMITHIES, Chicago, Ill.: I think that Dr. Ullrich's contention that this condition is congenital is well borne out by the direction in which the stomach herniates, namely to the right. That is the direction that congenital hernias of the diaphragm practically always take.

Dr. Hemmeter's point as to why such openings are not sewed up requires explanation. It is not a good plan to sew up any normal opening through the diaphragm, but to fix the stomach so that it cannot herniate through them. You remember the case that Dr. Basch reported, in which the autopsy was the most interesting feature? To differentiate between traumatic and congenital diaphragmatic hernia, one should remember that in a traumatic hernia the hernia is generally to the left. There is a direct break in the continuity of the diaphragm, and no alteration in any normal orifice. In these traumatic herniae, the operative procedure is to reduce the hernia and then sew up the diaphragmatic opening. This is always done in such cases, and the operation is successful.

The term "eventration" has been used. I think that it should be discarded. Such term means congenitally high-lying diaphragm on one side, in which the viscera lie abnormally high, but still in the abdominal cavity. There is no hernia in eventration. In the examination of traumatic herniae, there is a break in the diaphragm shadow locally while in congenital hernia, the diaphragm outline is continuous, and the movements equal on each side. In eventration, diaphragm continuity is maintained but practically always the left diaphragm lies higher than the right diaphragm and its motions may be sometimes limited.

CLINICAL REPORT OF A FEMALE (COLORED) PATIENT WITH TUMOR IN EPIGASTRIUM, A SECOND TUMOR IN LEFT BREAST—HEMETEMESIS—ABSENCE OF GASTRIC SECRETION, ETC., WHICH CASE DID NOT PROVE A CARCINOMA—RECOVERY.

By **John C. Hemmeter**, M. D., Phil. D., LL.D., Professor in the University of Maryland, 739 University Parkway.

BALTIMORE, MARYLAND.

Preliminary Remarks.

This is the patient who vomited blood shortly before she came into the hospital Friday, October 27, 1916, and who has a tumor in the left breast, another in the upper abdomen, and a chain of enlarged glands in the neck.

A number of possibilties were mentioned regarding the primary trouble. Among them, *first,* gastric carcinoma; *second,* an adhesive mass following a tubercular peritonitis, with a bunching of the omentum; *third,* a hypernephroma; *fourth,* an old indurated ulcer which might have perforated the stomach wall, but was walled off by a mass of omentum; *fifth,* syphilitic neoplasm; *sixth,* a wandering kidney.

A discussion took place in the clinic as to whether the breast tumor was a benign or malignant one, and also if malignant whether it was the primary or secondary growth.

To arrive at a diagnosis an examination of the kidneys, by injection, would decide the hypernephroma point, or that of floating kidney. A negative Wassermann would rule out lues. A culture of peritonitic fluid obtained by puncture would throw light on the factor of tuberculosis.

An X-ray series of the stomach after a meal of a glass of milk in which had been stirred one and one-half ounces of bismuth subcarbonate might diagnose cancer. The X-ray might also show bismuth remaining in the crater of an excavating ulcer.

The 'string test,' if positive, would indicate an erosion, either cancer, ulcer, or both. (Or if there was intense congestion with oozing.)

The examination of the stomach contents and of the feces might show blood, after a diet free from meat. This also would indicate erosions somewhere in the digestive tract or at the anal orifice if patient had been kept on a meat-free diet. After all of this had been done, examination of the eyes was made by Dr. Looper; the vaginal and pelvic examinations made by Dr. Hugh Brent; and the kidney functioning is reported on by Dr. Underhill. Dr. Arthur Shipley examined the breast tumor, and Dr. Carroll made the rectal examination. Dr. Walton's report on the X-ray series of photographs is also incorporated later in this report; along with the laboratory examinations we made of the blood, urine and feces, and peritoneal fluid and the anthropometric measurements.

Entrance Note on Admission: Patient's name, M. T.; chart No. 3663; ward "E;" bed No. 7; married; colored; female. 10/20/16.

Patient is a fairly well developed and well nourished colored girl about 25 years of age. Came into hospital today complaining of pains in abdomen. Patient states that these pains come on about one week before her menses, and that they are sharp and very painful. These pains are localized about her umbilicus and occasionally in epigastrium. Patient does not know whether she lost any weight but she states that she vomited a *large amount of blood* a few days ago. (One quart in red clots.)

History: Black, female, married, Dentsville, Md. Complaint: "Pain in stomach(?)" blood vomiting; blood in stool.

Family History: Father died with paralysis, age 65; mother dead, heart trouble, age not known. Four brothers living and well. One sister living and well. Five brothers and sisters dead—age and cause of each not known. No family history of malignancy, T. B., kidney disease, syphilis or insanity, no haemophilia.

Past History: Patient only remembers having had measles, otherwise a sound woman. Denies scarlet fever, chicken pox, smallpox, typhoid fever, malaria, rheumatism, diphtheria, T. B., pneumonia, tonsillitis, chorea, pertussis, etc., genito-urinary diseases.

Habits: Patient uses tea in moderation. Does not use any drugs. Denies use of alcoholics.

Venereal History: Denies Neisser infection and chancre.

Menstrual History: Patient was 16 years old before her menses began and they only came on after having taken medicine. Menses occurred every 4 or 5 weeks and lasted for 4 or 5 days, without pain. For last two years the patient had such severe pain that she had to stay abed. Lately one week before onset of periods she has pains about her umbilicus.

Present Illness: About 3 years ago following the birth of her last child the patient noted that she had some swelling of feet and ankles, marked dyspnoea and pains in her back and abdomen. The pains have persisted and the patient states she has become progressively weaker. For the last year she has been so weak that she has been unable to do anything. Patient has occasional attacks of oedema of feet and ankles.

Alimentary: Patient's appetite is very poor. She has occasional attacks of nausea and vomiting and the last time she vomited blood. No pains in stomach region. Marked eructation of gas. No fullness nor weight after meal. Patient has no aversion for meats or salts. Bowels are regular as a rule. Last week during stool patient noted that she passed blood.

Pulmonary: Patient has occasional attacks of dyspnoea. States she coughed up blood this past week. Denies chills, fever or sweats. States she thinks she has lost weight.

Cardiac: Occasional dyspnoea and oedema of feet and ankles, No palpitation. No pericardial pains. Valvular sounds normal.

Genito-Urinary: Nycturia. No polyuria. No urgency, frequency. Some tenesmus.

Nervous: No headaches. No dizziness or vertigo.

Special Senses: Patient states that she can not see clearly out of right eye ever since birth of first child. Dr. Looper's report is incorporated in another part of this paper.

Eyes: None of the signs of Basedow's disease nor hyperthyroidism. Thyroid gland not enlarged nor sensitive, no evidence of vagotonia nor sympathicotoria.

Physical Examination: Patient is a fairly well developed and well nourished negress, about 25 years of age. At time of examination is lying in the D. D. position, apparently suffering no acute pain nor discomfort.

Head: Is well formed and of normal contour, covered by fair growth of black hair. No scars, tumors, masses nor alopecia noted. Scalp is freely movable.

Eyes: Pupils are equal in size and symmetrical. React readily to both L. and A. Extrocular movement normal. No evidence of exophthalmos-nystagmus nor strabismus. Sclera of muddy color. Conjunctiva of clear white color.

Nose: Negative.

Ears: No tophi. No discharge. No tenderness over mastoids.

Mouth: Teeth are in a fair state of preservation. No pyorrhoea. Few decayed teeth. Tongue protrudes in midline without tremor. Slight whitish coat. Throat is markedly congested. Tonsils somewhat enlarged. No evidence of pus formation.

Neck: Well formed and symmetrical. No scars, tumor masses nor eruption noted. Thyroid is not enlarged. No tracheal tug noted. The posterior cervical glands on left side are distinctly palpable. Few glands in ant. triangles and at angle of jaw. No pulsation of neck vessels. No rigidity of neck muscles. No opisthotonos.

Chest: Well developed and well nourished. No scars, eruptions noted. Chest movements are symmetrical. Chest expansion normal. Respiratory rate normal. There is marked retraction of the supraclavicular fossae on the right side. No bulging nor retraction of the intercostal spaces. Percussion reveals impairment of the P. N. over Rt. Apex to 1st rib; otherwise percussion is negative. Auscultation shows roughened B. S. over Rt. Apex and distant B. S. over Rt. base posteriorly. Over rest of lung there is normal resonance present. No evidence of consolidation nor fluid.

Heart: Apex beat is in the 5th I. S., about 3 inches to left of M. S. L. No shock nor thrills noted. Sounds at apex are clear and distinct. No evidence of shocks, thrills nor murmurs. Sounds at bases are clear and distinct. No murmurs nor accentuations noted.

Breasts: Breasts are large and flabby. Left breast shows in upper right quadrant a tumor mass the size of a large egg. It is smooth, freely movable over the underlying tissues, attached to skin. Not nodular, not tender, not inflamed and does not show any fluctuation.

Abdomen: Is retracted. No scars nor eruption noted. Liver and spleen not palpable. Abdomen is very relaxed and shows striae of pregnancy. In the upper abdomen, just above the umbilicus, and a little further to the right than to the left is seen a *definite mass.* It moves up and down with respiration. On palpation it is felt as a hard, nodular, freely movable mass, slightly tender and about the size of a large egg. The mass is not attached to the abdominal wall, nor is it attached to the liver. It can be moved about readily. Kidneys are not palpable. No evidence of ascites. No tumors in pelvis.

Genitalia: Examination negative.

Lower Extremities: No scars nor eruptions. No evidence of oedema, varicose, nor tibial exostosis.

Reflexes: Knee and ankle—periosteal, radial and elbow reflex—Normal. Babinski and ankle clonus negative.

ENTRANCE NOTE, PHYSICAL EXAMINATION AND HISTORY BY DR. FRANK MARINO.

Anthropometric Examination: The patient has a well formed chest as mentioned in physical examination, and the X-ray plates will show that the stomach is in normal position and that the hepatic flexures and the splenic flexure of colon are fixed high up in their normal positions; she has not the appearance of a person who has misplaced viscera.

"Stiller's sign," or floating 10th rib is not present.

The anthropometric measurements are as follows:

Manubrium to Xiphoid	18	C. M.
Manubrium to Umbilicus	32	C. M.
Manubrium to Symphysis	45	C. M.
Xiphoid to Right Ant. Sup. Spine of Ilium	22½	C. M.
Xiphoid to Left Ant. Sup. Spine of Ilium	22½	C. M.
R. Sup. Spine of Ilium to Left Sup. Spine of Ilium	20½	C. M.
Circumference at Xiphoid	67½	C. M.
Sub-Xiphoid Angle	70°	

The 10th ribs are freely movable but are not detached.

Age, 23; sex, female; race, colored; weight, 120 pounds approximately. Patient states she has lost weight. Height 5 feet 5 inches.

Treatment: 50 grns. "Kaolin" to 1 qt. water. Lavage of stomach before breakfast. Fast for 1 hour.

Attempt to fatten patient by diet and hydrotherapy. Rest in bed.

HCl gtts 15-20 dilute in water after meals.

Kidney pad for movable left kidney.

Her peritoneum was tapped through posterior vaginal cul de sac for fluid in order to examine for T. B. bacilli and maculate a guinea pig with some of the fluid withdrawn—negative.

The patient had been sitting in erect position for 2 hours before the peritoneum was aspirated through the vagina, which was previously sterilized by solution H_2CL_2.

The following special examinations have been made, separate and distinct from the general examination.

Eye Examination: This was made by Dr. Looper on the 26th, and because of the fact that the patient had frequently complained that she saw but imperfectly from the right eye. It is as follows:

"Extra-ocular structures negative. Muscular movements good in all directions. Pupils equal, round and react freely. Vision each 20/20. OPH. Exam.—Lens and media clear in each. Optic discs normal. Peripheral fundus negative.

Impression—Eyes seem to be normal.

Rectal Examination: This was made by Dr. Carroll because of the possibility of finding a neoplasm. There was no hemorrhoidal condition or fissures. The anal canal was normal, nor was any growth found in the rectum. However, pieces of retained feces indicated an atomy of the rectum.

Gynecological Examination: Dr. Hugh Brent made this. He found no tumor masses present in the pelvis, but there was a badly lacerated cervix.

Breast Examination: Dr. Arthur M. Shipley carefully examined the tumor of the left breast, which has already been described and diagnosed it "intracanicular myxoma," with "cystic degeneration." He thinks that the slight scar over the tumor is the result of a local infection and has nothing to do with the tumor mass, *which is benign.*

Blood Examination: Dr. Maldeis made a Wassermann on the 21st and this was negative. Hgb. 80%, W. B. C. 9600, R. B. C. 4,220,000. The differential count—Poly. 72%. S. Lym. 23%, L. Lym. 2%, Large M. N. 2%, Trans. 1%. (This count might help rule out T. B.)

Blood Pressure: 120 Systolic—Diastolic 90—P. P. 30.

Sputum negative to T. B., few pneumococci—leptothorici.

Feces: Large stool, dark brown in color—normal odor. Occult blood—positive. Negative to parasites and ova. No segments of taenia saginata or solium. No free fat. Starch granules in small amount. No undigested meat fibres after Ad. Schmidt's diet.

Examination of Stomach Contents: Efforts to secure test meals with the stomach tube were only fairly successful because of the great amount of mucus, which was mixed with the food. Gastric contents were secured with a duodenal tube two hours after a single test meal had been given. The contents consisted almost entirely of mucus, which was streaked with red blood. No food debris was visible. The specimen was slightly yellowish in color and was odorless. There was no free HCl. acid present and there was no rennin, which was tested for as follows: An equal amount of filtered content was added to milk. Curdling had not taken place at the end of 10 minutes. HCl deficit=32. Proteolysis according to Mett's method=0. Second test HCl deficit=30.

Microscopical Examination: Many mucoid cells, numerous large mononuclear cells, no polymorphonuclear cells. The nuclei of the epithelial cells present were universally granular. We were not able to find the Oppler Boas bacilli. The absence of food residue can be explained by the rapid peristalsis as shown by the X-ray photographs. The diagnosis of this stomach condition might be "A mucus anacid gastritis," with "achylia gastrica." The bleeding may be due either to an erosion or as a result of intense congestion of the capillaries. There was no retching when the tube was introduced and it hardly could have come from injury in swallowing the tube.

October 28. Gastric contents again drawn 3 hours after meal. Blood again positive. Free HCl absent. Mucous present in large amount. HCl deficit 34. Milk not curdled in 2 hours.

String Test: This was done October 24th. It was positive to red blood in two places; one near the yellow portion of the string and the other just beyond where the yellow portion began. The length from the incisor teeth indicating a bleeding ulcer in the duodenum.

Urological Examination: The first examination of the urine was made on the 25th. Color, light amber; slightly cloudy, aromatic odor; Sp. gr. 1029, and at this time albumin was negative and sugar negative. The microscopic examination showed no casts and no pus cells. A later examination demonstrated blood and pus in the urine from the catheterized left kidney.

On October 27th Dr. Underhill catheterized the two kidneys and secured a specimen of urine from each kidney. Following this he injected the pelves of the kidneys with collargol.

X-Ray Examination of Kidneys: The patient was taken to the X-ray room and Dr. Walton secured an excellent pyelograph, which shows "both pelves normal, regarding outline." The kidneys are in normal position. The urine from each kidney was examined and macroscopically that from the left was darker in color and cloudy. That from the right was normal in color. Microscopically the left kidney urine shows numerous pus cells and R. B. cells. Urine from the right kidney normal.

Kidney Diagnosis: Pyonephrosis of left kidney and a L. kidney which is freely moveable, but which at the time the X-ray was taken had fallen back into its normal position.

On October 28th the left kidney was again back in normal position, but was manipulated until it reached the mid-line.

Dr. Maldeis failed to find T. B. in a catheterized specimen.

During the first palpation the left kidney had descended to the level of upper margin of the umbilicus.

X-Ray Report of Stomach Series: Four plates were taken following a meal 12 hours previously of one glass of milk mixed with ozs. 1½ of barium carbonate. The first plate was taken immediately after a repetition of this meal. The second one-half hour later. The third was taken an hour later and the fourth three hours.

The first plate shows: A slightly dilated stomach of a cow horn type, with a well filled cap, no filling defects but with pylorus somewhat to right of usual position and with pre-pyloric bulging. The previous meal is still in the caecum and ascending colon, where it remains throughout the series, evidencing stasis. The hepatic and splenic flexures are well up in propor position, the colon is redundant.

The second plate at one-half hour shows active gastric peristalsis well filled cap and bismuth progressing rapidly through the intestine.

The third plate shows stomach nearly empty and bismuth well along towards the caecum.

The fourth plate shows stomach completely emptied in three hours. The last meal is in the last part of the ileum.

The first third of the transverse colon appears in all plates approximated to the ascending colon and there is practically no progress of the first bismuth meal upward in the ascending colon.

Conclusions: An inflammatory condition in the upper right quadrant of abdomen with adhesions of colon and duodenum to liver.

CONCLUSIONS.

The definite findings are:

1st. A tumor in the breast; diagnoses as benign.

2nd. An X-ray series which indicates an absence of gastric carcinoma.

3rd. A positive finding of pus and blood from the left kidney, which appears to be a wandering kidney. The pelves of both kidneys are normal in capacity and outline.

4th. There was a positive string test.

5th. There was blood in the gastric contents.

6th. There was blood in the feces.

7th. Wassermann negative.

8th. Tubercle bacilli negative in peritoneal fluid.

On November 11, 1916, the tumor mass in the abdomen which was seen to move with respiration at the last clinic, disappeared. On manipulation in the left lumbar region it was again brought to the midline and diagnosed as a floating or third degree kidney. It was thought best to take a collargol X-ray. The vomiting of blood and blood in the gastric contents is hard to explain. She has none of the usual ulcer symptoms, such as hunger pain or relief of pain on the taking of food. The pain she had in the abdomen was near the umbilicus and at the sight of the tumor we saw. It did not extend through to the back and she had had no similar previous attack. The X-rays indicate abdominal adhesions in the upper right quadrant of abdomen. The breast tumor is not a metastasis, but is benign, cystic tumor. Her vision is normal in both eyes and the eye examination is otherwise negative, notwithstanding her complaint. The blood pressure is normal. The liver is normal in size and the heart is normal also, so that it would be hard to describe the blood in the stomach to a congestion following disease of these organs.

This unusual combination of signs and symptoms was interpreted as due to a gastric carcinoma when the patient was first presented at Prof. Hemmeter's clinic because of the hematemesis, achylia gastrica, abdominal tumor and blood in stool. The further study of this case revealed the nephroptosis, the benign nature of the breast tumor, the absence of lues or tuberculosis and a probable ulcer in the duodenum.

The treatment consisted of absolute rest in bed. Feeding through duodenal tube that was introduced so far as to rest in the beginning of the ileum. Later on iron and strychnia, abdominal bandage and support for the prolapsed kidney.

November 1, 1917, reported well and gained in weight.

Manuscript copied by Mrs. H. W. Miller, 538 N. Arlington ave.

DISCUSSIONS.

DR. HEMMETER: I do not know. I do not think she has a gastric cancer, because her recovery was permanent. It may be a case in which gastric or duodenal ischemia had caused bursting of capillaries. The case is doing very well, and I have presented it to show some of the didactic difficulties. I should be interested if I knew that other teachers had similar experiences. One of the shortcomings of our Association is that we have good discussions concerning classical cases or large compilations of clinical work, but none concerning clinical errors and didactic difficulties and none concerning the history of our specialty. If we had some papers of this kind at the banquet, for instance, we should be surprised to find how many things we had which individual members consider original have in reality been done by other investigators many years ago.

Dr. A. L. Benedict, Buffalo, N. Y.: During the Spanish-American war I had a patient, a young woman of twenty-five or thirty years, who was the wife of one of the soldiers in the service at that time. The patient was very much emaciated when I first saw her, and had various gastric symptoms. On passing the tube, an obstruction was found at the cardia, which was noted two or three times, and there was stagnation of the contents, showing more or less obstruction at the pylorus. Whether there is such a thing as a specific cancerous cachexia, or whether what we call cachexia is a generally run-down condition, this case had as type a cachexia as ever develops; it was suggested that an operation be done. The surgeon wanted to do a gastro-enterostomy, which I regard more as a vivisection than as a therapeutic measure. It was decided, however, that if the surgeon was called, he should resect the carcinoma or, if this were impossible, short circuit the obstruction by a vestecstomy. At operation tumors were found at both the cardia and the pylorus. Resection was not made. Intestinal fistula was established. After a time, on account of neurotic features, mouth-feeding was resumed, and the patient made a good recovery. She was alive the last I heard of her, so apparently the case was not cancerous.

I think that we occasionally get these cases that resemble cancer and prove not to be; and this shows the fallacy of the surgical argument that one hundred cases of carcinoma die under medical treatment to ninety under surgical; and that, therefore, an operation should be done in all cases. The real trouble, so far as diagnostic ability is concerned, is that we are dealing, not with a series of cancer cases, but with a series of cases that suggest cancer and many of which are not.

Dr. John A. Lichty, Pittsburgh, Pa.: This paper of Dr. Hemmeter's shows nicely the advantage of thorough examination of the patient, and also the advantage of so-called team-work, which we are speaking of now-a-days quite frequently in connection with diagnosis.

The hemorrhage in this case is, of course, the point which has not been fully explained. Probably the following paper of Dr. Austin will throw some light on this, because, I think, arterial change, such as may go with certain conditions, may manifest itself through a hemorrhage in the gastrointestinal tract. The other point in the paper, that is, the tumor of the abdomen, which was seen at one time and then disappeared, only to reappear subsequently, is interesting.

Now a movable, or floating kidney, is the one tumor that is most likely to exhibit such phenomena. During the past year, I saw a patient who was sent to me by Dr. Ziegler, of Pittsburgh. The patient had a tumor in the abdomen which was thought to be a movable, or floating kidney. He was able to push it away up under the costal margin, but it reappeared. We had a genito-urinary man inject the kidney and radiograph it, and found that the mass was not the kidney, which was shown to be in the normal place.

The patient had been operated on before, to shorten the ligaments of the uterus. I had been telling my students for a number of years that after an abdomen had once been opened, one should discount all the signs and symptoms by about fifty per cent. as you are very likely not to be able to depend on them so thoroughly as you could in an abdomen that had not been opened.

At operation we found a very unlooked for condition. When the previous operation was done, the omentum was found attached in the region of the cecum. There had been an old appendix trouble, and it had been tied off with a ligature. This ligature was not absorbed, but became encysted, and an abscess had formed about it, producing the movable mass. Pus in this abscess sac was absolutely sterile.

I refer to this case in order to show that an abdomen that has once been opened does not subsequently give signs and symptoms that can be relied on the same as before being opened.

Dr. Jerome M. Lynch, of New York: I might mention in this connection a very interesting case referred to me about two weeks ago which shows the necessity of being very guarded before reaching a conclusion of basing a diagnosis on X-ray examination alone. This woman had a large mass in the pelvis and after a very thorough examination combined with X-ray a diagnosis of carcinoma of the sigmoid was made. A proctoscopic examination revealed an enormous fecal mass which was removed piecemeal after several peroxide enemas.

Dr. Hemmeter: What became of the tumor?

Dr. Lynch: I removed it with peroxide of hydrogen. It was an enormous fecal impaction.

Dr. P. E. Truesdale, Fall River: The case which Dr. Hemmeter reports presents an interesting picture, and the problem in that case is to explain the cause of the hemorrhage. Inasmuch as it is always difficult to define the character of a tumor that is movable anywhere in the abdomen, another guess in regard to that might also be in order.

A patient was sent into our clinic with a diagnosis of peptic ulcer, and the history was somewhat cloudy. There was an area of tenderness in the epigastrium, and a tumor could be defined, which was at times in one place, and at times in another. The patient was somewhat anemic, had lost in weight, and had had these periodic hemorrhages, and the assumption was that it was probably a malignant growth, but the blood-picture was rather interesting. It showed some evidence of a splenic anemia. There was a leukopenia, and consequently our attention was directed towards some disease of the spleen. On opening the abdomen, the tumor, which was floating in the upper abdomen, was found to be an enlarged spleen, and this explained the whole picture. The hemorrhages from the stomach and bowel were apparently caused by the condition of the spleen.

I assume that this possibility was taken fully into consideration in Dr. Hemmeter's case. Mine was a case of Banti's disease.

DR. HEMMETER, closing: I did not report the case in detail, because I did not think that it was desirable to do so, owing to the brevity of the time.

The blood examinations were made. I wish to say that this is an observation that I have made repeatedly in purulent vomiting. I first studied it in a sea voyage between here and Italy, which was very tempestuous. I am not a good sailor, and after vomiting three days, the vomited material was found to be streaked with blood in my own case. On other sea voyages, I have noticed that the patients who vomited severely for more than forty-eight hours are very apt to vomit blood eventually. I have no other explanation than the word ischemia for that, because it passes off as soon as the patients reach the shore.

In this case, owing to the displaced kidney, the patient had vomited a good deal before I saw her, and I was inclined, purely on assumption, to think that the hemorrhage from the stomach was due to the vomiting, which stopped as soon as the kidney was in position. All of you have probably seen this occur when there was twisting of the ureter and displacement of the kidney. I thought that this was the proper explanation, because of the patient's rapid recovery and the negative result in other organs in the abdomen.

ANGINA ABDOMINI.

By Edward H. Goodman, M. D.,
PHILADELPHIA, PA.

American Gastro-Enterological Association, May 1, 1917.

The term angina abdomini was introduced by Baccelli to describe painful seizures in the abdomen due to aneurysmal formations in the vessels of the celiac axis, or due to arteriosclerosis of these same vessels. Another name for the same condition has been offered by Huchard angina pectoris pseudogastralgique. Inasmuch as the name invented by Baccelli has been accepted and used by recent writers, notably Pal, Brunton and Williams, Minella, Breitmann and others, it is the term which should be used in the future for this disease.

Patient's History: The patient, a well preserved woman of 82 years, apart from trivial illnesses of but short duration, has enjoyed good health for the greater part of her life. Twelve years ago she suffered with intense pain in the stomach which was diagnosed at this time carcinoma ventriculi. In view of the present state of health the diagnosis is subject to question. Disregarding this attack, which resembled the seizures recently experienced, the patient rejoiced in the best of health and spirits until the early part of September, 1916. Following hard physical work connected with house cleaning, a task which no appeal to regard her years could make her depute to others, she was stricken with violent and intense pain in the umbilical region, which was accompanied by great anxiety, nervousness, restlessness and attempts at expulsion of gas per rectum and ex ore. These attacks having no relation to meals, were repeated frequently, medication and diet proving unavailing, since September. She was seen by a surgeon November 19, as gall stones were suspected, but the diagnosis was not confirmed. Operation was disadvised, but no satisfactory conclusion was reached concerning the nature of the attacks. The surgeon's examination, which was most thorough, failed to reveal any organic lesion other than the arteriosclerosis.

The patient was counseled to remain in bed, to partake nothing except milk, and was given a mixture of hydrochloric acid, and pepsin, with occasional doses of sodium bicarbonate and nutmeg. For three days she was so much better that her physician permitted her to get out of bed, and again with utter disregard for her years, she cleaned a closet and laid, unassisted, a heavy carpet (November 23), being alone in the house at this time. Following this exertion she was seized with an excrutiating pain in the umbilical region, with restlessness, extreme nervousness, attempts at expulsion of gas, no abdominal distention, no passage of urine or feces, some nausea, but no vomiting. The following morning this attack was duplicated in all characteristics except that the pain was less formidable. I saw the patient in company with the attending physician approximately about four hours after this second, and to date, last attack. The seizure on this day had been of short duration, as were all the others, that is to say the acme of the frightful pain was sustained for only a few minutes, but intense abdominal soreness followed, and was present when I saw her, and lasted for a period of about twenty-four hours.

I found the patient (November 24) to be a remarkably well preserved old lady, of frail build. The pulses were equal, regular, normal in rate, but the vessels were hard and easily palpable. The heart examination revealed a faint systolic murmur at the base which was interpreted as being caused by aortic roughening.

The stomach was slightly dilated, there was a splash (milk had been ingested but a short time before), there was some diastasis of the recti. The abdomen was very sensitive to pressure, particularly in the umbilical region corresponding to a circular area about six inches in diameter, having the umbilicus as its center. There was marked throbbing of the abdominal aorta, no evidence of any enlargement of the vessels, and no murmurs. The reflexes were normal and the lower legs were slightly edematous. A rectal examination made by the surgeon five days previously and being reported negative, was not repeated. The urine has contained albumin, no sugar and no casts. The blood pressure was not taken at this time, as I had neglected to take my instrument, having been told it was essentially a stomach case. My neglect in this respect I have since regretted, as my request to the attending physician for a subsequent report of the blood pressure was overlooked. However, four months later the pressure was found to be 130-60.

I believed we were confronted with an almost typical case of angina abdomini, and although sceptical as to the correctness of this view, the physician consented to give my therapeutic recommendations a trial. To the patient was explained what we believed to be the cause of her distressing attacks, and we gained her support in the view that physical over exertion or nervous strain, or both, were predisposing factors in calling an attack into being. Therefore the recommendation to rest during the day, and particularly a half hour's rest on the right side with a hot water bottle to her abdomen following her meals, was readily agreed to. She was permitted to eat anything sparingly at the regular hours for meals, and a glass of milk with a biscuit between meals. Only meat was restricted. Nitroglycerin gr. 1/100 was given every three hours, and potassium iodid gr. X three times a day. Perls of amyl nitrit were left, in the event of a recurrence of the pain.

Since November the 24th the patient experienced the beginning of an attack, but the attack was cut short by the prompt use of amyl nitrit and since that date there have been no further attacks. Potassium iodid and nitroglycerin have long since been discontinued, the patient is active in Red Cross work, and seems to be enjoying the best of health, the fear of cancer having to her great relief been permanently dispelled from her thoughts.

The symptoms as disclosed by the history of this patient seem almost typical of what has been described by others as angina abdomini and a further discussion of the symptomatology of a classical case would seem unnecessary. Permit me to recall the cardinal facts. The main feature of this woman's illness was the sudden agonizing pain which was directly associated with physical over exertion or nervous shock, thus in a way resembling the onset of angina pectoris. There is a prevalent notion that all attacks of angina pectoris are determined by emotional or physical effort, yet from Heberden himself we learn that attacks may occur during sleep and often during rest. Diderot records an attack which aroused him from sleep (Allbutt). Nevertheless, many cases of angina pectoris are precipitated by exertion, and in this patient of mine, effort seems to have been the exciting factor. The pain is described by the patient as being horribly severe, and after the first seizure, the dread of another, and the realization that others were to occur, since the attacks were repeated, made her life one of continuous apprehension.

Accompanying the pain there were efforts at expulsion of gas by mouth and rectum, and when belching could be effected there seems to have been some slight relief. The bowels were slightly constipated. There was slight nausea but no vomiting.

The situation of the pain was about the umbilical region and radiated to the back, and folowing the seizure there was great sensitiveness on slight pressure about the umbilical region for an area six inches in diameter.

The diagnosis should not be difficult, and to my mind the main support of the correct diagnosis should be based on the frightfulness of the pain, the short duration of the same, the occurrence of the pain in arteriosclerotic individuals, usually advanced in years, in whom, apart from the arteriosclerosis, there can be found no appreciable sign of disease. Men are said to be more frequently affected than women.

The differential diagnosis must, in some cases, be made between abdominal angina and other diseases associated with gastric crises. Here it may be stated that Allbutt believes that epigastric angina is but a somewhat aberrant form of ordinary angina, and due, broadly speaking, to disease of some part of the thoracic aorta, but in angina "more definitely abdominal," he thinks it may be due to disease of the lower portion of the aorta or in other large vessels there. Osler is disposed to discredit the diagnosis, regarding abdominal angina as but the abdominal symptoms of angina pectoris. In this patient of mine, despite her years, no symptom of any cardiac disease had been complained of. There were no transient seizures without pain in which "there was a general suspension of the minor operations of nature for three or four seconds," called angina sina dolore.

Abdominal aneurism should not be a matter of much moment in the differential diagnosis, as the pain is never anginoid, but rather boring in character, and situate in the left abdominal region. (Of 179 cases reported by Nixon, none had anginoid pains.)

Tabes dorsalis should be readily distinguished, as the reflexes point the way to the true diagnosis, unless, unhappily, the patient is a sufferer from both tabes and from angina abdomini. The pain in tabes is intercostal rather than abdominal.

Closure of the mesenteric vessels, I mention as an improbable differential diagnostic difficulty on account of its extreme infrequency and because of the impossibility of recognizing the condition unless one has been the observer of a previous case. Closure of the mesenteric vessels may take place in either the veins or arteries, as a result of thrombosis or embolism. For thrombosis arteriosclerosis itself is an underlying predisposing cause, for embolism valvulitis is the contributing factor. The onset of mesenteric closure is sudden and leads rapidly with great pain to collapse. Bloody vomitus is seen, and the stools, which contain blood, may be either obstipated or diarrheic. The abdomen becomes distended, painful and resistant to touch, with dulness in the dependent portions of the abdomen. The temperature is normal, pulse is rapid and soft and the patient gives the impression of one suffering with severe peritonitis or intestinal obstruction. In no case should abdominal angina be mistaken for mesenteric closure.

I should prefer to leave the question of pathology undiscussed as but few cases have been studied satisfactorily from the clinical and pathological sides. Various suppositions have been suggested, but "clinical and pathological precision has yet to contribute to a better analysis of these phenomena." (Allbutt.)

As far as therapy is concerned, rest is an important measure, and avoidance of physical or nervous strain is to be recommended. Diet is to be that generally recommended for individuals of advanced years. Massage and faridization of the abdomen have been advised, and the homely hot water bottle is not to be despised. Potassium iodid, sodium nitrite, sodio theobromine, salicylate, nitroglycerin, amyl nitrit, may each have its place, alone or in careful combination. During an attack, if amyl nitrit fails, morphine hypodermically may be employed.

248 South 21st Street.

DISCUSSIONS.

Dr. J. C. Hemmeter, Baltimore: I think that one should not express any emphatic opinions on a case like that without having seen it personally. The symptoms correspond well to those of a patient whom I have seen, off and on, for two years, in which it was a very difficult problem to decide whether the patient had angina pectoris, with the pain essentially referred to the solar plexus, or a very advanced case of locomotor ataxia. That the Wassermann test is not always positive in that condition is now a well known fact. This is due to the difficulty in getting a satisfactory test in syphilis of the central nervous system. Why this difficulty exists, I do not know.

I should like to call attention to the phraseology. The word angina has no reference to pain. It comes from the Latin word angina, or the Greek word 'αγχιονη and means to throttle, to choke or suffocate, and if you wish to express a condition in which pain is the predominant symptom, the word angina, it appears to me, is a misnomer, because it does not mean pain. Pathologically it means CO_2 accumulating in the blood and resulting in suffocation.

Dr. G. W. McCaskey, Fort Wayne, Ind.: The case of Dr. Goodman opens up a very interesting and important field. It is a little premature, perhaps, to attempt any acute analysis of this subject, until we know a little more about the pathology of angina pectoris itself, a subject on which we are very ignorant at the present time. I have seen several cases somewhat like Dr. Goodman's and I am inclined to think that many, and possibly most, but I am not willing to say all, are really cases of angina pectoris with the pain referred to the solar plexus. In one of my patients, in whom there was frank cardiac disease, of which the patient finally died, there were attacks of pain, always referred to the abdomen. In ordinary angina pectoris we often have no physiological signs of cardiac disease. There is another group in which the disease may be lower down, but I think myself that it is an open question. The fact that the attacks come on with exertion, seems to me to point to the cardiac mechanism as the site of the real pathology. Angina pectoris, as quoted by the essayist and as we all know, does not always occur upon exertion. I remember distinctly one case that came to autopsy with coronary arteries like pipe stems—a typical case, in which more of his attacks occurred in the middle of the night than at any other time. They would come on from exertion, but they commonly came when he was sleeping soundly. That was undoubtedly a case of what has been called ordinary angina pectoris, and the cardiac lesions demonstrated at autopsy. When in the hospital, he had indigestion, to which I did not attach much importance, and sent him home finally, and later, I got a report from the doctor that his stomach symptoms had increased, and he died of gastric cancer, entirely unsuspected by me. In fact, he had ordinary indigestion, which one may expect in severe cases of cardiovascular disease, so I feel a little non-committal on the whole question of pathology. While there may be a type in which the lesion is located in the abdominal vascular apparatus entirely, and for which, according to Dr. Hemmeter's criticism, which is obviously correct, we should have some other name; yet I think that it is a fair question, in these cases, whether the cardiac mechanism itself is not the primary cause.

Dr. Jacob Kaufmann, New York City: Without going into a discussion of what the term angina really means, I think that the question at issue here is, Are there attacks of severe pain in the abdomen evidently caused by disease of the abdominal arteries—attacks of severe pain that are similar to the attacks of pain as we see them in true angina pectoris caused by diseased coronary arteries? I would answer that question as positively, Yes. It is perfectly true, that in certain cases of true angina pectoris the pain is located in the abdomen, as Dr. McCaskey indicated; just as it is true that in other cases severe pains are located in the chest, thus simulating angina pectoris, when as a matter of fact we are dealing with an abdominal process which provokes the pain. I have seen cases of gall-stones, which, for a long time, went under the diagnosis of angina pectoris; and in which the pain ceased with removal of the stones. The question more precisely then is, Are there such attacks of severe pain occurring in the abdomen caused by arteriosclerosis or other disease of the abdominal arteries? The only proof can be had when you get the finding, at autopsy. I had occasion to such an observation, later published by Dr. Schwyzer, with whom I saw the case. The patient, a middle-aged man, was subject to sudden severe attacks of abdominal pain, always associated with most pronounced gas distension of the stomach. He finally succumbed during such an attack, and at post mortem was found an almost complete obliteration of the arteria gastrica of luetic origin. While the wall of the stomach was comparatively unchanged. Here then we had proof that in this case the pain was caused by a periodical complete closure of the artery with consecutive paralysis of the gastric wall, producing that enormous gas distension.

Similar observations are reported by Ortner, who describes cases in which pronounced arteriosclerosis of the mesenteric artery led to severe attacks of abdominal pain, also with consecutive paralysis of the gut, simulating intestinal obstruction. Several cases were operated on; and at the post mortem, conditions in the mesenteric arteries were found similar to those which we had in our case of obliterative sclerosis of the arteria gastrica. I think that these are cases of true angina abdominali. While it may be a risky thing to make the diagnosis, still it is on the same basis as we have the occurrence of pain in the lower leg in cases of intermittent claudication with arteriosclerosis of the arteria tibialis; and in cases of intermittent lumbago which according to Hunt occur when the arteries supplying the muscles of the back are diseased. As in these cases, the pain is provoked through the exertion of the involved muscles, so in the abdomen the exertion is caused by the digestive process or by general exertion in combination with that, leading to the closure of the diseased arteries.

Geo. M. Lockwood, New York City: It seems to me that, aside from the cases of angina abdominalis characterized by severe and paroxysmal pain, we have two other groups that must be included in the same category, which may throw some light on the pathology of the affection. Both are rather minor groups. One is that group in which the patient complains of recurrent abdominal pain or distress during the height of digestion, depending rather upon the quantity of the meal than its quality. The second is the type in which the patient experiences abdominal or epigastric pain if he exercises after eating. Some of the patients with pain when exercising on a full meal, if they stop for a while, can go on again without pain; just as in chronic aortitis. In other cases, the pain recurs as soon as they begin again to exercise. When we consider what is probably going on in the stomach, we are led to surmise that these cases are due to a diminished blood supply of the stomach. During the height of digestion the blood vessels are as full as they can be; and if the tension is raised by exercises, it is too much for the arteries and we have a condition analogous to intermittent claudication, and of a similar pathology. I think that these cases of angina abdominalis are real entities. I think it is very difficult, however, to make a differential diagnosis between these minor cases and those cases of aortitis with substernal or retrosternal pain, associated with aerophagia (?), with distension of the stomach, causing epigastric distress.

DR. JOHN P. SAWYER, Cleveland: I wish simply to make a statement regarding the pathology of these cases. A year and a half or so ago, one of our colleagues was taken ill, at 4 A. M., with intense pain in the abdomen. The physician gave a hypodermic. The patient rested for a time. I got there at 7:30, and he was just having a fresh attack of intense pain. After brief examination and consultation I stepped to the telephone to call a nurse; but before I got the nurse on the telephone, the man was dead. Post mortem gave the typical findings of a tremendous arteriosclerosis of the abdominal aorta. The picture which one ordinarily sees in the examination of the coronaries of the heart in these extreme cases was magnified into the dimensions of the aortic proportion, without anything else being found whatever to account for it; and it is a striking instance of angina abdominalis with a pronounced aortic lesion.

DR. GEO. W. MCCASKEY, Fort Wayne, Ind.: May I add just a word? This discussion has been along the line of an assumption which has been questioned; that is, that the pain is like intermittent claudication, and due to cardiac-ischemia. I do not think that the picture is nearly so clear as that. It is true that in most cases, perhaps, we have a real stenosis of the coronary arteries; but there are cases in which this is absent. As I remember this theory of intermittent claudication is now generally discredited to some extent, in explanation of the symptomatology of angina pectoris.

In regard to the Wassermann, I think it is unfortunate that this test was not made. Dr. Hemmeter referred to the difficulty of getting it in the central nervous system. My cases practically all have it; and the place to find the test is not in the blood, but in the spinal fluid. If the patient has luetic disease of the central nervous system, I will get it there. I am making more lumbar punctures for the purpose of clearing up the condition. A large majority of the cases of lues of the central nervous system do not have positive blood Wassermanns.

DR. FRANK SMITHIES, Chicago: I think that there is such a thing as angina abdominalis, but I wish to give a word of warning against expecting to find a high systolic blood pressure. We may have very marked general sclerosis without high systolic blood pressure; and in just such cases as Dr. Goodman has mentioned. Here the cardiac factor is weak and we have low systolic pressure. I remember a case of the sort in which the systolic pressure was 110 and the diastolic 80. That there is a circulatory factor is evident, because the patients often die of heart failure. The nearer the aortic pressure approaches the intra-ventricular pressure, the sooner is cardiac death to be expected; so that we cannot say that the patient has not angina abdominalis and may not be suffering with symptoms of such, because there is low blood pressure. Dr. Goodman states that the blood pressure in his case was low. That was the systolic, which I consider is the least important pressure to take. The diastolic pressure in an aged individual is of far greater importance.

I would also call attention to another upset that may closely simulate these cases. Dr. Goodman's case had diastasis recti. We had cases in which he had epigastric hernia. A small part of the gut was pulled in and pinched, and closely simulated, in an arteriosclerotic, what might be called angina. In another case we had small gravel-like gall-stones which intermittently passed through the cystic duct. There was no evidence, to the surgeon or the X-ray man, of stone; so it could readily have been overlooked. The passage of a small stone may closely simulate cases of "abdominal angina."

Dr. Goodman, closing: I am very glad to have the discussion center about the pathology. The gentlemen who discussed my paper have described cases similar to my own, but I was disappointed that they did not speak of the effect of amyl nitrate, which was of great benefit in my case. I agree that arteriosclerosis is of no diagnostic significance, and also agree that arteriosclerosis may be present with a low systolic pressure. The question or the suggestion that the bowel is pinched in the condition of diastasis recti is interesting, but I do not think that there was any pinching in my case. I do not deal with the prognosis of this condition, as one cannot speak authoritatively from a single instance. According to other writers prognosis seems very favorable if the patients follow the ordinary rules of hygiene and diet.

Dr. Henry W. Bettmann, Cincinnati, Ohio: The condition under discussion is illustrated by a patient I saw last fall, a woman of nearly 80, whose case was closely analogous to the one Dr. Goodman reported. The patient lived in a town in southern Ohio. During a visit to Washington she had severe attacks of colic and notwithstanding her age the consulting surgeon decided to operate for gall-stones, which, however, were not found at the operation. The attacks of pain were renewed on her return home, and on examination we found all the signs of advanced arteriosclerosis. I think it would probably be wise if we all published our cases of angina abdominalis, in order to save other patients from useless surgical interference. The symptoms so closely simulate gall-stones in some cases that, unless we bear in mind the possibility of angina, we are apt to have our patients operated on.

A DIAGNOSTIC SIGN OF GASTROENTEROPTOSIS.*

BY CHARLES D. AARON, Sc. D., M. D.,
Professor of Gastroenterology in the Detroit College of Medicine and Surgery.

DETROIT.

Gastroenteroptosis is easily recognized by the use of the Roentgen ray, inflation, auscultatory percussion and transillumination. The author wishes to urge an additional method in diagnosis which suggests a correlative form of treatment.

In cases of gastroenteroptosis, deep, continuous pressure with the ends of the fingers, over the celiac plexus, in the epigastrium will induce pain. (Fig. 1.) The point of sensibility varies in different individuals. To locate it, the co-operation of an assistant is necessary. With the patient standing, the physician applies his fingers in a series of deep pressures until the point of greatest tenderness is found. The fingers are held at this point. The nurse then takes a position behind the patient, passes both arms about him so that the hands, meeting in front, rest on the hypogastrium and lifts the abdomen in its entirety. (Fig. 2.) This relieves the epigastric pain at once, despite the great pressure

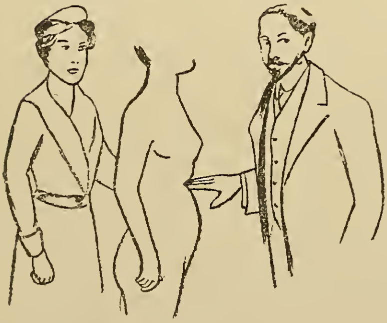

FIG. 1.

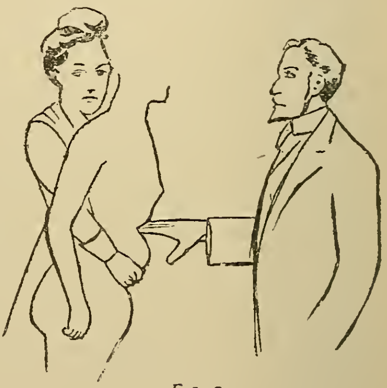

FIG. 2.

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*Read before the American Gastroenterological Association at Atlantic City, April 30, 1917.

exerted by the physician at the point of tenderness. When, however, the nurse allows the patient's abdomen to drop to its former position, the deep pressure continuing, the pain reappears. (Fig. 1.)

This sign is constant in gastroenteroptosis. In organic disturbances, such as gastric ulcer, carcinoma, etc., the pain under pressure continues even when the abdomen is lifted.

Glénard made clear the necessity of supporting the abdomen by means of a bandage in cases of gastroenteroptosis exhibiting discomfort when the hands supporting the abdomen were suddenly removed. My method confirms this view by the fact that the sensibility to pain through pressure ceases when the abdomen is manually lifted, and equally calls for the employment of a supporting bandage.

Glénard believes that the epigastric pain under pressure indicates a displaced or diseased liver. But sensibility to pain is not always within a definitely circumscribed region; it may be of a diffuse nature. Lennander declares that the intestine, whether normal or diseased, is insensible to pressure, and that pain, when present, is produced through hyperesthesia of the parietal peritoneum. Kelling, again, explains pressure pain by irradiation or reflex diffusion through the abdominal nerve plexus. It is also known that pain may arise from a hyperesthesia of the nerve plexus surrounding the abdominal aorta. The epigastric point of pressure is especially pronounced in nervous derangements of the stomach. My observation, however, leads me to the belief that the pain provoked by the pressure of the fingers upon the epigastrium in cases of gastroenteroptosis is due to the fact that there is a tugging on the nerves in the mesenteries, due to the weight of the abdominal viscera, irritating the celiac plexus. Elsner remarks that the sensibility to pressure pain has its seat in the sympathetic ganglion and nerve plexus of the abdomen; he also believes that the stretching of the nerves in the mesenteric causes pain at the point where the diseased organ connects with the nerve plexus.

The pain on pressure, relieved by lifting the abdomen, reveals an objective sign which I believe to be reliable in the diagnosis and treatment of gastroenteroptosis.

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DISCUSSIONS.

Dr. Charles D. Aaron, Detroit: I have found this sign valuable in the case of patients who would refuse to wear an abdominal bandage though in need of one. When you can show them that by lifting the whole abdomen, with both hands on the hypogastrium, you can relieve them of their pain, they are willing to wear the bandage. This procedure has helped me very much in the treatment of these cases.

Dr. Henry W. Bettmann, Cincinnati, Ohio: I did not hear the first part of Dr. Aaron's paper, but I do not think we need any help in diagnosing the actual existence in gastroenteroptosis. If, however, the elevation of the abdomen will temporarily relieve the pain or cause it to disappear, that is a positive indication that the patient would be benefited by wearing an appliance. That is something we cannot tell so immediately by any other test. I think that, from that point of view, Dr. Aaron's contribution may prove valuable.

CHRONIC INTESTINAL STASIS WITH RESPECT TO CERTAIN ETIOLOGIC FACTORS.

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CHICAGO, ILL.

During the past ten years there has been much surgical enthusiasm over the operative relief of colon stasis. It has been difficult for the physiologist or the internist not to believe that much of the interest of the surgeon lay in the successful mastering of intricate and novel operative maneuvers. In the fascination of performing operations of the type suggested by Lane, it would seem that not infrequently the question of ultimate benefit to the patient has occupied a minor position.

Ochsner has quite well defined the type of case in which surgical procedures for the relief of intestinal stasis offers the least disappointment. His experience in our clinic at Augustana Hospital indicates that rather more than one-half per cent. are permanently benefited by colon removal, with or without short-circuiting. It would seem that in the selection of material for either surgical or medical procedures, it is quite necessary to consider the means at our command for demonstrating (1) the clinical proof that stasis exists, (2) that with this stasis there *is* a toxemia, and (3) what surgical or medical measures are available for the alleviation of one or both the above conditions.

1. *Proof of the Existence of Stasis.*—As a rule, it does not require a medical diploma for one to be able to determine the existence of delayed evacuation of the bowel in a given individual. The patient usually makes the diagnosis himself and resorts to a physician only when common household remedies have failed. That many individuals who are constipated are of the physical type described by Lane is well known. It has been the common knowledge of both doctor and layman for centuries. There are no definite statistics at hand, however, that actually demonstrate that the constipation class of the human

family is made up largely of sweaty, clammy, sallow, glum-looking, feebly-acting people. On the other hand, it is well within the clinical experience of all that many patients who conform physically to the "Lane type" have normally acting bowels and that numerous instances of obstinate constipation present themselves where the individuals are rosy cheeked, even plethoric, and mentally alert. It should be recalled, when considering the clinical type popularized by Lane, that many of these individuals have passed the time of life when exuberance of circulation and of spirits is logically to be expected. Many of these patients have had severe damage done to the body economy by disease and not a few have experienced mental traumata. Moreover, the group upon which surgical operations are performed is essentially a picked group—it includes persons who are, as it were, in the *last stages* of a type of distress associated with faulty evacuation of the bowel. Naturally, when statistics are collected regarding the type of individual associated with intestinal stasis, the surgical group upon which operations have been performed is composed largely of the "Lane type" individuals. It would seem to me that it is impossible to say that there exists clinically a distinct type of individual, which type has *resulted* from intestinal stasis alone.

Symptoms of Intoxication Due to Intestinal Stasis.

1. *Constipation.*—The most constant symptom is, of course, the persistent constipation which may, however, be interrupted at times by diarrhea, caused directly by irritation in the colon as a consequence of accumulations of hardened fecal material. This last condition is always late in its appearance, and only after constipation has existed for a considerable period of time.

2. *Malnutrition.*—Patients who are suffering from chronic intestinal stasis are often badly nourished. The condition reduces the appetite and thus the amount of food ingested, and because it seemingly impairs the digestion, and the patient does not obtain the normal amount of benefit from the food taken.

3. *Skin Changes.*—The complexion of patients suffering from intestinal stasis may lose its clearness, the skin may become yellow with brown spots, dark areas may appear beneath the eyes and there may be added pigmentation of the skin in the axilla, the groins and other flexures. But this is by no means the rule.

4. *Impaired Circulation.*—In some instances the circulation is below par. Such is manifested by the presence of cold hands and feet and sometimes by a syanotic condition of the skin. The patients may complain of shortness of breath upon attempting severe muscular exercise or upon mounting stairs.

5. *Impaired Muscular Strength* renders these patients unable to perform their accustomed amount of physical work. They become exhausted easily and do not recuperate quickly upon taking rest.

6. *Impaired Resistance.*—Intercurrent diseases are borne badly as the normal fighting power seems at times to be reduced.

7. *Effect Upon the Nervous System.*—These patients often appear greatly depressed nervously. They may develop melancholia or hysteria. It is, of course, important to determine which has been the primary condition.

Many neurotics develop intestinal stasis as a secondary condition, their neurotic condition having had some other foundation. The "intoxication" resulting from the intestinal stasis increases the unfavorable symptoms in these cases and treatment for relief of intestinal stasis usually improves their general condition. However, unless this "intoxication" is the sole cause, such individuals soon become worse again and quite commonly, considerably worse than they were before the treatment was begun, especially if this treatment has been surgical in character.

Lane and many of his disciples give other symptoms, and some authors have attributed practically every possible complaint to "toxemia" caused by intestinal stasis. No doubt this toxemia has a harmful effect upon the physiologic functions of every organ of the body, and by placing great weight upon this one item a clinician of susceptible mentality can read its influence into the history of almost every patient. Just as the dietician or the climatologist can find causal relations between disease of every form and the subject of his especial interest, so can the practitioner who is an enthusiast in the study of intestinal stasis apply his fad energetically, notwithstanding there are frequently obvious medical or surgical anomalies of a tangible nature present.

8. *Faulty Habits.*—Too little attention is paid to children developing a systemic regime in emptying the bowel. This is not infrequently due to carelessness on the part of parents. As has

been shown by physiologists, the ingestion of food furnishes a normal stimulus to the act of defecation. In the lower animals, dogs, cats and horses, etc., prompt evacuation of the bowel usually follows feeding. In normal infants it has been observed by nearly every mother that either breast or bottle feeding is usually succeeded by a bowel movement. When children get old enough to run about, the normal stimulus of feeding with respect to bowel movements is often ignored. After meals children usually hurry off to school, out to play or are occupied with "home-work" or other labors. This leads to retention of material for a long time in the colon and to irregular evacuations. The retention of inspissated material in the lower bowel is not by any means an unimportant factor toward producing colonic distension. This habitual colonic distension undoubtedly weakens the muscle tone of the colon and leads to permanent dilatation of the colon with attendant harmful retention of contents. As Franklin White has shown, retention in the colon especially about the cecum, the ileo-cecal valve and the terminal ileum, have a definite defect toward producing gastric upsets, especially those upsets associated with pyloric spasm and delayed gastric emptying. Too early in life a child cannot learn the value of going to the stool systematically, particularly at times when there is a physiologic stimulus to evacuating the colon, as, for example, after breakfast or after the evening meal. If these habits are carefully established in early life, undoubtedly many instances of chronic constipation would be avoided and much damage to the wall of the gut.

X-Ray Demonstration of Stasis.—With a very few exceptions, actinologists are quite prone to consider an intestinal function entirely at fault when an opaque meal administered by mouth is not completely evacuated by the natural channels in forty-eight hours. Jordan, working with Lane, has been particularly active in claiming that the X-ray method was of valuable clinical service in showing intestinal stasis, Jordan's published report of his technique of examining this type of case, definitely states that the opaque motor test-meal is administered to stasis cases without the bowels having previously been emptied. In an individual who presents himself at a clinic for the relief of a constipation, where in many instances evacuations from the bowel have not taken place for days or even weeks, it is quite difficult to see how one can expect an artificial meal such as is used by roentgenologists, to pass from the intestinal tract within anything like "normal time." From what we know of barium or bismuth compounds physically and chemically, it is difficult to understand how such substances can either permeate material already blocking up the lumen of the bowel or how these can work their way past inspissated fecal material and later be discharged. It is not astonishing that in practically all cases of constipation that are examined by the X-ray method as outlined by Jordan, plates demonstrate retention of the opaque meal for varying lengths of time. It would seem to be somewhat miraculous, if in an individual whose colon is already packed tight with dried material, a heavy metal compound such as bismuth, *could* work its way out of the intestinal canal. In our experience in determining the patency of the intestinal channel, particularly of the colon, it has been shown that only reliable information can be obtained when the motor power of the gut is determined by a meal administered by mouth, after the intestinal canal has been thoroughly cleaned out twenty-four hours previously.

That the commonly employed opaque meals *are* physiologic, has not yet been proved by even the most enthusiastic actinologists. Pawlow, Cannon, Carlson, and others have repeatedly reported experimental work which proves how important psychically, with respect both to secretion and motility, are those impressions conveyed to individuals or animals as the result of a meal appealing to the special senses. Sight, taste, smell and even color all contribute to proper gastro-intestinal activity. To anyone who has had personal experience with the way patients regard opaque roentgen meals, it is not necessary to explain that the feeling toward such is one of non-interest at least. To many, the meals are disgusting. This being true, it is impossible to regard the evidences of stasis as shown by the X-ray method as being entirely reliable. Even if the special senses were satisfactorily catered to by opaque roentgen meals, it must not be forgotten that a large quantity of bismuth or barium is from its nature, chemically, not wholly inert in the intestinal canal. It is well within the experience of all of us that one of the common remedies administered to control diarrhea is bismuth. Whether such bismuth has its effect as a sedative to an irritated intestinal lining, or whether it kills infecting organisms or it neutralizes poisons, it is impossible to state. The clinical fact remains, however, that commonly diarrhea is stopped after the administration of bismuth. It does not seem altogether clear why we should expect large volumes of bismuth administered to an ailing patient to pass through the bowels as does a meal that the patient himself has chosen according to his own desires and which excites the secretions of the gastro-intestinal canal and which stimulates a physiologic motor apparatus.

It should also be remembered that by whatever food medium bismuth is suspended in the motor meal, such food medium is commonly called upon by the digestive enzymes of the saliva, gastric juice, pancreatic juice, bile and succus entericus. Consequently what may have been a perfect suspension of the opaque meal when taken by mouth very early loses its efficiency as the mass travels along the gastro-intestinal canal and as a consequence shortly after the opaque meal leaves the stomach the large volume of barium or bismuth is no longer in suspension but exists as an inert mass—foreign body—which excites no physiologic stimuli in the alimentary tract and which has a weight out of all proportion to its volume.

In our clinic, we have definitely shown that such a thing as "normal time" for opaque meals to pass through the intestinal canal is a term which demands wide interpretation. Quite commonly, individuals in perfect health (orderlies, elevator boys, nurses and the like), to whom barium or bismuth compounds have been administered by mouth, showed retention of the opaque meal in the colon from three to five days. In these individuals no evidences whatever pointed to constipation or intestinal stasis. On the other hand, we have instances where patients came in with obstinate constipation, but evacuated the motor meal within forty-eight hours, or with very slight delay, if previously the intestinal canal had been freed from old retention contents. Moreover, in both normal and constipated individuals, we have shown that if the same individual be examined at *different* times, there is a wide variation in the hours it takes the opaque meal to pass through this gut.

During the past two years it has been our custom to estimate the rate of onward progress of food through the intestinal canal by the administration, with ordinary food, of such inert non-diffusible materials as carmine or lycopodium. The coloring matter is administered with food of the patient's own choosing, in a small capsule, and note is made of the time when stools are passed, stained with the dye or mixed with the spores of lycopodium. Sometimes it is difficult to feed a hearty meal to a chronically constipated individual. The majority of these patients are mal-nourished, inasmuch as after years of constipation they feel that harm is done to the body economy by the ingestion of meals which they either know or think they know will not be later evacuated. This factor of chronic mal-nourishment may be a not negligible one in bringing about a chronic pernicious form of ailment in an individual who, initially, was affected with but a temporary upset. When a coloring matter is administered in the above manner, results are quite striking. In our experience the majority of patients who claimed that intestinal stasis of the obstinate type existed, were demonstrated to pass stained stools well within the time limit of people with normally acting bowels. In but a few cases were unstained stools not recovered within thirty-six hours after the administration of the coloring matter. Not uncommonly, in these obstinate cases, an enema revealed that stained material lay in the sigmoid and rectum awaiting evacuation and such material could frequently be discharged spontaneously by the patient upon regular effort. It would seem that the above simple method for determining the presence or absence of actual stasis offers possibilities in determining the presence or absence of actual stasis offers possibilities in determining the selection of individuals for different forms of therapy.

We have elsewhere already emphasized very graphically that opaque enemata are not to be relied upon too greatly for the indications that stasis exists when plates show an apparent malposition of the colon. In our experience it has been shown that the colon may occupy a multitude of positions following the administration of opaque enemata and that the clinical evidences of malfunction bear but a slight relation to the position that colons occupy as shown by the X-ray plate. Moreover, in a given individual it is not infrequently possible to secure pictures indicating wide variation in the position of the colon when plates are made in series. These variations in position can well be observed if studies are carried out with the aid of the fluoroscopic screen, while enemata are being given. They can be well noted and profitably studied after the administration of large doses of atropine hypodermatically. Not only does the position of the colon change, as shown by a series of plates, but very often angulations, twists, and kinks can be demonstrated which appear to have particular significance only if they are looked to as causative factors in the production of an existing obstinate constipation. Few such abnormalities can be shown to be persistent in the average "stasis" patient examined. We have already shown that the demonstration roentgenographically of "incompetent ileo-cecal valve" bears absolutely no relationship to the clinical picture of stasis, so-called. We have numerous instances where markedly "incompetent ileocecal valves" have been shown in connection with chronic constipation, chronic diarrhea, or absolutely normal stools. The physiologists have frequently informed us that there is a "physiologic incompetence of the ileocecal valve." If plates happen to be made at the time that the valve is open, regurgitation of the opaque meal into the lower ileum may be expected in any type of case, clinically. While certain uncorroborated chemical and bacteriological studies of the contents of the lower ileum have been reported by Mutch from instances of intestinal stasis, it does not seem to us that roentgenographically there is any evidence to indicate that "intestinal stasis" really means "ileal stasis." Lane, who has the largest experience in handling cases of chronic constipation, finds the fewest instances of incompetent ileocecal valve.

Of course, it is granted that when organic causes of stasis are present (tumors, diverticula, adhesions, stenoses from chronic ulcer, and the like) roentgenograms are of valuable service in accurately localizing the seat of the lesions. However, in this type of case, definite clinical manifestations of mechanical hindrance to the onward progress of bowel contents are but rarely lacking and the clinical history is commonly characteristic.

2. *Proof of the Existence of a "Toxemia" Dependent Upon Intestinal Stasis.*—I have already commented upon the variability of the clinical appearance of chronically constipated folk. Although Lane and his co-workers have laid strong emphasis upon the usual biochemic and bacteriologic findings in the gut (particularly the terminal ileum) of individuals who came to operation on account of intestinal stasis, these observers have never produced by the administrations of these "poisons," in experimental animals or humans, the general or local anatomic

defects which, dogmatically, they claim result from fecal stagnation and retention. Among bacteriologists and chemists, there is much skepticism regarding the possibility of poisons or abnormal bacteria passing from the lumen of the gut directly into the systemic blood or lymph streams. It should be recalled that the bowel wall is not merely an inert animal membrane filter, whose "porosity" varies according to laws, purely physical. The contents of the gut are separated from the circulating body fluids by a *viable* animal membrane in which complex biochemic processes are constantly being carried on. All dependable experimental evidence indicates that even were "split proteids," ptomaines, aromatic compounds, soluble bacterial toxins or pernicious micro-organisms constantly present in abnormal quantities of the bowel affected with fecal retention, such inimical substances rarely pass unaltered through the wall of the intestine. Thus far, scientists have generally shown that a chief function of the gut is to render such harmful material inert. No evidence is forthcoming that establishes the fact that where intestinal "stasis" exists, the normal biochemic functions of the bowel wall are permanently altered and that this alteration could be directly ascribable to the stasis itself. It is quite likely that preceding stasis some not yet understood damage may have been done to the bowel wall which damage permits of abnormal interchange between the circulatory streams and that the stasis itself may be but a result of this underlying fault.

I have been frequently impressed with the history of chronic undernourishment and chronic infection in many of our stasis cases. This undernourishment or the history of infectious ailments have not rarely long antedated the history of chronic constipation. Many of these patients have been previously affected with infectious ailments of the mildly or viciously active septic type and in the majority of these individuals local foci of infection can be demonstrated if carefully searched for. Decaying teeth, pyorrhea alveolaris, infected tonsils, sinuses or lymph glands, and, upon laparotomy, diseased appendix, gall-bladder, fallopian tubes and the like, are common findings. In most of the cases, the clinical evidences that these foci of infection existed long before the onset of chronic constipation are readily established. I feel that this association of chronic partial starvation particularly in females) and the presence of germ centers in some part of the body, cannot be too strongly emphasized with regard to the subsequent onset of intestinal stasis. In all stasis cases, not only should the *history* of previous infectious diseases be ascertained, but careful search should be made for the *persistence* of local infected foci. That such foci, in the presence of faulty feeding, fatigue or irregular habits, may become extremely harmful, has been demonstrated by Woolley and by Olmer. The latter has shown that when such opportunities are given, bacteria multiply very rapidly. The increase can be actually proved by culturing the venous blood. Improvement in the general body state is accompanied by prompt sterilization of the venous blood.

Examination of sections of the large bowel that have been extirpated surgically in cases of chronic intestinal stasis, usually show areas of muscular atrophy or even patches of scar tissue. These zones are not infrequently in more or less direct relationship to bands of peritoneal surface. Not infrequently the colon is actually bound down or constricted by these evidences of old healed inflammatory processes. At points of the large bowel where stagnation and bacteriologic activity are marked, these old scars may actually produce fixation of the bowel. Such are very often seen at the cecum, hepatic and splenic flexures and about the sigmoid. It has been supposed that many of these bands or veil-like envelopes of the large bowel were congenital. It is quite likely that such represent extension of inflammatory foci, from the lymph channels of the large bowel itself. In instances where scars or muscular atrophy have not been observed, the muscle bundles forming the wall of the large gut are frequently swollen, edematous and present all the appearance of a chronic myositis, both grossly and microscopically. It is quite likely that persistent attempts at tissue culture from the walls of the colon will disclose actively infecting organisms such as we now commonly recover from the appendix, gall-bladder or stomach walls. It would appear that in many instances the initiation of a chronic intestinal stasis is due to an infective myositis of the wall of the large gut. This may be locally, in numerous small patches, or quite generally distributed in large areas. The effect of such inflammation is apparently similar to that where a myositis exists in other parts of the body, namely, there is interference with free muscular activity, imperfect strength of contraction and definite prevention of consecutive peristaltic rhythm. Under such circumstances, bowel contents are propelled onward without orderly peristaltic sequence. This doubtless permits accumulation of gas and feces in segments of the bowel, which accumulations act as local irritants or produce distention of the bowel with muscular stretching. Such conditons as these not infrequently incur as complications of the *acute* infectious ailments—typhoid fever, pneumonia, etc., resulting in abdominal distention and painful peristalsis. In such ailments the effect is, however, but temporary and recovery is the rule. If, however, a large bowel muscle is constantly being infected over wide area by slowly growing or intermittently active organisms, which tend to congregate in small groups, then the damage to the intestinal wall is long maintained and constant new bacterial traumata are being inflicted. Such bacteria not uncommonly are fed to the digestive tracts from chronic infections of the tonsils, teeth, head sinuses and doubtless from numerous intra-abdominal foci, as, for instance, the appendix, gall-bladder, ulcerated surfaces in the stomach or duodenum or from the pelvic organs. It would thus appear that local foci of infection, particularly if such are closely connected with the digestive tract, have a not altogether unimportant influence upon the subsequent development of a type of imperfect bowel function which is associated with infrequent evacuations.

3. *Proof that Surgical or Medical Measures are Available for the Treatment of Chronic Intestinal Stasis and Its Associated So-Called Toxemia.*—No clinician of experience will deny that whatever means are used to aid this unfortunate class of patients, the task is a difficult one. To alter their psychic state alone is a problem. Certainly early attempts should be made to rationalize these folk. Not rarely, the elimination of their fads is as difficult as is the elimination of their intestinal contents. The best results from treatment come from the earnest co-operation of patient, surgeon and internist. The internist must seek surgical aid in eradicating all local infected foci, wherever in the body such may be situated, before he can successfully begin procedures of non-surgical type. If the stasis persists after a reasonable interval when such details have been attended to, then laparotomy *should always be exploratory in the fullest sense of the term.*

Very commonly, the removal of an infected appendix, a gall-bladder filled with stones, a constantly recurring peptic ulcer, a foul uterus and its adnexa or the separation of gross bands of adhesions will render quite unnecessary the uncertain "short-circuiting" operations. The last type of procedure should be avoided whenever possible, for in a given case, at the time of laparotomy, one can never prognose what future functional state will exist following the short-circuiting. Even when the surgeon has done a perfect job mechanically, patients very frequently later present themselves to the internist on account of obstinate and distressing diarrhea or for constipation of a degree exceeding even that experienced before operation. Not only can anomalies of the bowel be then demonstrated, but in some cases I have been able to show that definite diminution has taken place in the digestive *quality* of the secretions of the stomach mucosa, the liver and the pancreas. Such alterations generally resist treatment, and then the woe of the patient is as great as is the perplexity of his medical attendant.

DISCUSSIONS.

Dr. Jerome M. Lynch, of New York: It seems paradoxical that the physician should be the advocate of surgery and that the surgeon should decry it. It is unnecessary to defend Mr. Lane or the operations he has suggested. As he was a pioneer in this branch of surgery it is but natural that it had not reached perfection nor does it follow because failure resulted from ileo-sigmoidostomy in many cases that colonic surgery be entirely abandoned. Dr. Draper and I believe that there are certain cases where right-sided resection of the colon which we have called developmental reconstruction is very definitely indicated. This is often in those cases of extreme angulation usually on the right side and which we have designated "elbow deformity." Some might contend that the simpler operation of cutting and pleating might accomplish the same result. This we have tried and in the majority of cases the improvement has been temporary. Some are satisfied to merely separating the bands, trusting in Providence that a recurrence will not take place. Resection is the well recognized procedure in all extensive traumata of the gut.

The fundamental principles underlying peritoneal asymmetries are imperfectly understood and as a consequence much confusion exists as to the proper procedure to follow while operating. Every band and adhesion is apt to be looked upon as a Jackson's veil due to an imperfect understanding of Jackson's original article. It is imperative to discriminate between delaminated peritoneum and ordinary inflamed adhesions. One may be the result of another, but until delaminated peritoneum becomes an adhesion it should not be cut.

Dr. Pfahler has mentioned elongation of the sigmoid as being due to neglect on the part of the child to go to the toilet. This seems inconceivable! Is it not a fact that the cecum and sigmoid are two of the most variable organs in the body and that the constipation is an effect rather than a cause?

THREE CASES OF COLON INFECTION.*

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Infection by the colon bacillus has been known for a long time. It is not uncommon as the infecting agent in the course of appendicitis, peritonitis, or of the commoner infections of the genito-urinary tract. As a general disease, however, there is little reference to it in the literature. The following cases have been observed, and although in some respects the data is incomplete, it has seemed to me worth while to put them on record. There are certain points of similarity about them that are of interest. The patients were all trained nurses and, as such, possibly more exposed to colon infections than others. In all, there was a persistent, but otherwise inexplicable fever; and in all, the diagnosis was made by the agglutination test, the blood cultures being negative throughout. In other respects, however, the differences were sufficiently marked not only in clinical manifestations, but also in the response to treatment.

Case I.—Miss W. G., *æt.* thirty, gave the following history: Rather delicate as a child and fainted frequently. In 1914, she had tonsillitis followed by polyarthritis and trifacial neuralgia. The attack was very mild and lasted about a week. Subsequently she had had some pain in the region of the heart, and had suffered from shortness of breath upon severe exertion. Her appetite was very good, her digestion was poor and she suffered from gas and eructation. Before she was taken sick, her blood had been somewhat anemic. A count made in February showed hemoglobin 84 per cent., red blood cells 3,940,000, white blood cells 14,500. She remained at work and in good health until August, 1916, when, deciding that she was too fat, she started to reduce by dietetic measures that included large amounts of lemon juice before breakfast and the omission of all starches and sugars from her diet. In one month, she lost 15 lb., and at this time developed heavy, aching pains in the legs that came on rather suddenly. Pains were also felt in the right side and in the muscles of the back and neck. At that time a physical examination was made and it was found that she had a pulse of 132. There was no enlargement of the heart to percussion, but a loud systolic murmur was heard over the pulmonary cartilage. The leucocytes were 19,800. The temperature was found to be 100° F.+. The urine contained acetone, diacetic acid and 31 per cent. of sugar—the total quantity of sugar passed in a day was 3.53 grm. The abdomen was tender, particularly marked areas being found above McBurney's point and just above the umbilicus. There was no tenderness over the gall-bladder. Her weight was 140 lb. She was immediately sent to the hospital and kept under observation. The temperature varied between 100° and 103° F., was distinctly irregular and remittent. There was marked tenderness constantly above the umbilicus, but the tenderness over McBurney's point disappeared. The leucocytes remained elevated. Sugar was present during the first few days of her stay, but soon disappeared. She was very nervous and restless and suffered a good deal from dyspnea.

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*Read at the annual meeting of the American Gastro-Enterological Association, April 30, 1917.

A diagnosis of subacute pancreatitis was made, and she was seen in consultation with Dr. Jopson; but we agreed that an operation was not indicated then, although it was not certain that it would not be necessary later. Various tests were made in order to determine the cause of the fever. The blood cultures were all negative. Urinary cultures were negative. Agglutination tests were made with the typhoid bacillus, with two strains of the paratyphoid, and with stock colon bacillus. With the latter, a strong agglutination was produced. With the others, the results were negative. It was thought that perhaps the colon bacillus would agglutinate readily with human blood, and it was tested with the blood of fifty-four cases in the wards suffering from various conditions. In one case, a slight agglutination was obtained. In all the others, it was negative. This seemed to show that we had not to do with a particularly susceptible organism.

The patient was placed upon rest, and a carbohydrate-free diet. As a result of the colon bacillus test, a vaccine was prepared with the reacting strain, and the patient given increasing amounts, starting with 100,000,000 and rapidly increasing to 2,000,000,000, injections being made every third day. The improvement was very marked. After the third injection, the temperature reached normal and remained there. All the subjective symptoms disappeared. Sugar, diacetic acid and acetone were no longer found in the urine. The injections were continued at longer intervals for about two months, and the patient made a complete and satisfactory recovery and has resumed her professional duties.

Briefly summarized, a young woman, with a slight tendency to obesity and a history of a previous mild general infection, as a result of a rather injurious diet, developed pain in the abdomen, fever, leucocytosis, and glycosuria. A diagnosis of pancreatitis seemed very likely. The cause of the pancreatitis was presumably the infecting agent, the colon bacillus. On vaccine treatment, combined with careful dieting, and the administration of pancreatic substance in glutoid capsules, the patient rapidly recovered.

Case II.—Miss E. E., a nurse, also *æt.* thirty, was referred to me by Dr. William Jackson Merrill. She complained of pain and stiffness in the joints, especially in the back. The previous history is rather interesting. She has suffered from two nervous breakdowns, characterized by insomnia, loss of appetite and anemia. She has had two attacks of pneumonia. In 1911, her appendix was removed and also dilatation and curettement for painful menstruation performed. Subsequently, there have been several attacks of quinsy, and in 1914 the tonsils were removed and dilatation and curettement was again performed; and in the fall of this year, an exploratory laparotomy was done, but nothing found. She has suffered from headache which was relieved by suitable glasses. She has had various atacks of diarrhea associated with fever. In 1913, she began to have pain in the left sacro-iliac joint. This pain then extended to the knees, shoulders and arms. Although it was fairly constant it produced no interference with motion and she has been able to attend to her duties. There has been no cough, no night-sweats and only slight shortness of breath upon exertion. The physical examination was negative. Her weight varied from 105 lb. to 110 lb. and has never been more than this. She has a temperature every evening ranging between 99° and 100° F. Her energy was considerable. In addition to her position as head nurse in the hospital, she perfected herself as an organist. The blood, blood-pressure and urine were all normal. The examination of the gastric contents was negative. Von Pirquet and Wassermann tests were made and were negative. Blood cultures were also negative, and finally a series of agglutination tests were made with various strains of micro-organism. Of these only the colon bacillus reacted. A vaccine was prepared and was administered at intervals of about one week in increasing doses up to 3,000,000,000 for a period of three months. The diarrhea was distinctly improved, but no other effect was noticed. There was no gain in weight, no alteration in the daily temperature, and no improvement in strength. It was suspected at this time that there might be some lesion of the vertebral column on account of the backache. X-rays were made by Dr. H. K. Pancoast, but they showed nothing abnormal. Dr. Merrill made a careful examination of the back and fitted a brace, but the brace could not be worn and Dr. Merrill reported that there was no evidence of spinal disease. X-rays were also made of the teeth and sinuses, as it was assumed that the removal of the appendix and careful exploration of the abdomen by Dr. Anspach had excluded the possibility of any abdominal infection. These were also negative. In addition to the vaccine, the patient has had various forms of treatment, including salicylates, iodides, local treatment to the colon, intestinal antiseptics, and pancreatic substances in glutoid capsules, etc. No effect of any kind has ben produced on the case excepting the improvement in the diarrheal condition.

This case belongs to the category of obscure, persistent fevers. The focus of infection has not been found and yet no inconsiderable efforts have been made to discover it. The nature of the infection is probably the colon bacillus, but treatment with vaccine prepared with a strain that agglutinated strongly with the patient's serum failed to have any definite effect. As to the nature of the condition, speculation is idle. There has been nothing to indicate the existence of a diverticulum, or chronic pancreatitis, or gall-bladder disease. The genito-urinary tract is apparently normal. The fever still persists, rarely, however, reaching 100° F., but usually exceeding 99° in the evening, and the patient's condition remains about the same.

CASE III.—Miss A., a trained nurse, has been nursing a series of mental cases for several years and was supposed to have had a nervous breakdown. The symptoms were sleeplessness, extreme apprehension, nausea, vomiting and vague pains. She tried to rest for several weeks without medical supervision, but seemed steadily to get worse. When I first saw her, she had fever and vague and shifting tenderness in the abdomen. The bowels alternated between constipation and diarrhea. The patient was highly emotional, weeping and laughing without much provocation. The leucocytes were low, two counts, each 6,700, being made at an interval of about a week. With our knowledge of the other two cases, blood tests were made early and the reaction with the colon bacillus was strongly positive. A vaccine was accordingly prepared and administered for a period of two months without any effect upon the fever or upon the symptoms of the patient. In the meantime careful X-ray studies were made, and, in the opinion of Dr. Newcomet, they indicated the existence of a chronic appendicitis. As tenderness could occasionally be elicited over McBurney's point, and as our medical treatment had failed entirely to relieve the patient, an operation was advised and accepted. The appendix was rather long and there were some adhesions about it but no definite inflammation. The report from the laboratory was chronic appendicitis. Nevertheless, after its removal, the patient made a prompt and complete recovery. Subsequent studies of the blood gave a persistently negative agglutination test for the same strain of colon bacillus. It may be noted that the gastric contents were normal. The feces were normal, although no cultures were made from them. The blood culture was negative. After the operation, the patient had a severe attack of acidosis with the presence of a trace of albumin and some casts in the urine. This lasted for three days.

Briefly summarized, it appears that this patient was suffering, probably for some time, with chronic appendicitis. As a result of this, she had a general infection with various gastro-intestinal symptoms and fever. As soon as the appendix was removed, she recovered from all these symptoms and has since then, for a period of six months, remained perfectly well. Vaccine treatment with strains of the colon bacillus had no effect whatever upon the course of the case, neither increasing nor diminishing the febrile process nor the subjective symptoms. With the removal of the appendix, the agglutination test ceased to occur. Why, after prolonged infection, the power of agglutination with the colon bacillus should have disappeared from the blood, it is difficult to say.

Some years ago, Dr. Joseph Evans and I reported a case of general colon infection in which chronic appendicitis was ultimately found. That this is not the invariable cause of colon infection may be inferred from the fact that one of the cases here reported had had her appendix removed and that in the other there was never any evidence of appendicitis and that complete recovery took place. It is more difficult to understand the effect of treatment in these conditions. In one case, the vaccine apparently was of great benefit. It may be, however, that the patient was beginning to improve when the vaccine was given and that this improvement would have continued if no interference had been undertaken. Impressions are very doubtful evidence in medicine, but it is my impression that the immediate effect of the vaccine was quite distinct and that it hastened the patient's recovery. In the other two cases, vaccine therapy, although continued for some time, was of no value. It must be remembered that the cultures were not autogenous. Nevertheless, the vaccine was prepared with the strain that agglutinated readily with the patient's blood. One serious defect in these studies was the failure to isolate the strain of colon bacillus from the feces, in order to determine whether it would react with the patient's blood. Blood cultures in all of the cases were negative.

DISCUSSIONS.

Dr. Jacob Kauffmann, New York City: In the cases of colon infection, was there anything found indicating an actual state of inflammation of the mucous membrane of the colon?

DR. MORGAN: Is there any discussion of the second paper of Dr. Sailer? If there is none, I will ask Dr. Sailer to close the discussion.

DR. SAILER, closing: The most careful study was made of the feces, with negative results in all three cases. No pus was found; no fermentation; no excess of white blood cells. So we are at a loss on that point. I am inclined to think that the cases were due more to general than to local infection.

Dr. Hemmeter's point is well taken. I should not have used the word perforation. It was a small opening between the two loculi of the stomach.

DR. MORGAN: Dr. White has some announcements to make.

THE ROENTGEN RAYS IN THE DIAGNOSIS OF INTESTINAL STASIS.*

By George E. Pfahler, M. D.,

PHILADELPHIA.

There is probably no greater authority and no one more capable of defining intestinal stasis than Sir William Arbuthnot Lane,[1] who says: "By chronic intestinal stasis I mean such an abnormal delay in the passage of the intestinal contents through a portion or portions of the gastro-intestinal tract as a result in the absorption into the circulation of a greater quantity of poisonous or toxic material than can be treated effectually by the organ whose functions it is to convert them into products as innocuous as possible to the tissues of the body." With this definition clearly before us we can easily understand that a patient may not have a bowel movement for three or four days or as many weeks and still retain health, because when such a person is healthy it means simply that a quantity of toxic materials absorbed from the retention is no more than can be treated effectually by the organs whose function it is to convert them into products that are innocuous. On the other hand, a patient may have a bowel movement every day or several times a day and yet suffer from intestinal stasis.

I have frequently had patients tell me that their bowels move like clockwork and that they have a bowel movement daily at a regular time of day without the assistance of any laxative or purgatives. The patients, however, had symptoms of intestinal stasis and when examined by means of the roentgen rays it was found that even though they had a bowel movement every day at a regular time and without purgation, at times the bowels would not empty themselves completely of a given opaque meal in four days.

Saundby[2] says "fecal retention is not inconsistent with a daily action of the bowels," and Hertz[3] emphasizes this same fact when he says "it is well known that the frequency of the stool gives no certain evidence as to the existence of intestinal stasis."

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*Read by invitation at the annual meeting of the American Gastro-Enterological Association, Atlantic City, April 30, 1917.

Hertz says "it is at all uncommon to be told by a patient, who is in the habit of taking large quantities of aperient for constipation, that the lassitude, headache and abdominal discomfort from which he suffers are ameliorated if he forgets to take his aperient and his bowels are not open." His symptoms appear to be due to the absorption of poison from the abnormally fluid contents of his colon, and are therefore a result of the diarrhea produced by his aperient and not a result of his constipation. It is important before examining such cases with the X-ray that the patient should not take any aperient for some days. When this has been done, however, I have often been able to demonstrate the complete absence of intestinal stasis, although many of the patients were contemplating some operation for the relief of their supposed constipation.

Effects of Intestinal Stasis.

In general the effects of the intestinal stasis are a toxemia, and Lane[4] believes that this toxemia can produce degenerative changes in every single tissue of the body. Johanbroeke[5] says "the intra-intestinal pressure from stasis may cause pain, inflammation, ulceration, and perforation of the intestinal walls, bodily and mental lassitude, increased nervous irritability, despondency, and acute insanity have been known to supervene." Bainbridge[6] says "chronic intestinal stasis is increasingly being accepted as one of the fundamental causes of disease." Metchnikoff[7] says "the ill health which follows the retention of fecal matter is certainly due to the action of some of the microbes of the gut."

Satterlee[8] says "the effects of intestinal stasis are rightly divided into mental, physical and nervous. Simple and seemingly isolated as these appear to be, it is evident with our recent knowledge that we have failed properly to understand their significance and to interpret their meaning. What multitudes of sufferers from neuralgias, myalgias, neuritides, hemicranias, neurasthenias, amauroses, all of which are now being treated for the symptoms rather than for the cause, and yet how little we know regarding the greatest problem of the relation of this subtle yet powerful toxemia to the more serious and better defined lesions of the cardiovascular system, central nervous system and genito-urinary system, which some of the more advanced observers consider to be reflexions of this fundamental bowel perversion. In general it cannot be too strongly emphasized that every patient presenting any of the simple symptoms, above mentioned, should be looked upon as essentially toxic until proved to be otherwise."

Reed[9] finds a constant relationship between intestinal stasis and epilepsy, and says "nearly two thousand X-ray plates in my possession attest the constant relationship of mechanical constipation to that form of chronic convulsive toxemia that is usually spoken of as epilepsy."

Great therapeutists have always recognized the influence that this toxemia may have upon any disease under treatment, and it is their rule always to empty the bowels as a first step in whatever treatment they are about to institute. Even the laity in general have recognized the influence this stasis or intoxication has upon the general system, and it has become a general custom to take a purgative no matter what the group of symptoms from which they are suffering. It must be our aim, however, as physicians and scientists, not simply to confine our efforts in recognizing the effects of this toxemia, but to go beyond and learn the cause of this chronic condition and so far as lies in our power to find the remedial measures therefor.

Symptoms of Intestinal Stasis.

The symptoms of intestinal stasis have been best formulated by Chapple,[10] who reviewed 50 cases in which he found the following constant set of symptoms: 1. Headaches, which were severe and frequent. 2. Attacks of nausea often followed by retching or actual vomiting. In some cases the vomiting was very frequent, indeed, and of a severe nature. It was very interesting to note that in several of the cases blood was frequently present in the vomit, and had led very able men to the diagnosis of gastric ulcer, although none such was found at operation. 3. Loss of appetite was almost constantly present. 4. Loss of weight was present in all cases. 5. Markedly cold hands and feet which gave an indication of a defective circulation. 6. Mental apathy; this was very definite in most cases. 7. Constipation, which in all was persistent and the previous treatments had failed. One case has gone on several occasions for twenty-eight days without an action of her bowels in spite of treatment. 8. A constant foul taste in the mouth, which was often accompanied by foul breath and carious teeth. The tongue was constantly furred. 9. Attacks of abdominal distention which were accompanied by discomfort and relieved by eructation, or the passage of flatus, or an action of the bowels. 10. General muscular pain and a loss of freedom in the movement of the joints. The physical examination, according to Chapple, reveals (1) skin staining as a rule of a sallow, brownish nature associated with more definite localized patches, especially under the eyes, in the axilla, and in the groin; (2) breast changes simulating, in the early stages, chronic mastitis and in the later stages, cystic degeneration; (3) abdominal tenderness over the areas of fixation.

The Diagnosis of Chronic Intestinal Stasis.

The effect of the toxemia upon the nervous system and upon the tissues of the body can be and should be recognized by the attending physician from the clinical symptoms and the physical signs, but the actual retention, the location of the retention and the cause of the retention can only be recognized by a careful roentgen examination. By this means we not only obtain the direct evidence of the cause of this toxemia, but we can determine the presence or the absence of many of the pathological conditions which give symptoms that may be confused with chronic intestinal stasis, as Lane believes, are the effects of chronic intestinal stasis, such as marked atheroma of the arch of the aorta, duodenal ulcers, gastric and duodenal ulcer, gastric carcinoma, gall-stones, carcinoma of the gall-bladder, disease of the pancreas, carcinoma of the bowels, etc.

Technique.—Briefly the study of the gastro-intestinal tract for intestinal stasis is accomplished by the administration of a barium sulphate meal, and this meal is observed fluoroscopically and roentgenographically by means of the roentgen rays in its passage from the mouth to the anus. Additional and very valuable information is also obtained by means of opaque enemata as observed fluoroscopically and roentgenographically. When this investigation is being made with special reference to intestinal stasis there should be no preceding purgation or starvation, *i. e.*, the patients should not be especially prepared for the investigation, but should be allowed to take their usual laxative as has been their practice for weeks, months or years, and they should be allowed to eat their normal meals and go about their normal duties so far as is practicable.

By this procedure one is not disturbing the conditions under which the symptoms have developed, and one will obtain a truer picture of the actual pathological state than can be obtained when an unusual procedure, such as purgation and starvation, precedes the examination. In general, however, the great majority of patients, who are sent to me for abdominal study, have a group of vague abdominal symptoms which might be accounted for by a gastric, duodenal, gall-bladder, appendiceal lesion or intestinal stasis. To study such patients ideally one should first carry through a careful investigation of the gastro-intestinal tract with reference to the intestinal stasis, and then, after thorough elimination of the barium sulphate, one should carry out the procedure of investigating the other abdominal organs. The expense and time consumed, however, makes this procedure impractical in most cases, and in this large group it is my custom to order for the patient a bottle of citrate of magnesia at 9 o'clock preceding the examination, and to report for examination at 9 o'clock the following morning without breakfast. Under these conditions the gastro-intestinal tract should be empty and quiet. This preparation is especially important for the study of the gall-bladder, because (1) if the bowels are not thoroughly emptied, confusing shadows produced by opaque or semi-opaque material in the colon, may lead to a false diagnosis of gall-stones; and (2) if the bowel is not thoroughly emptied it does not permit the study of the lower right side of the abdomen for gall-stone, which is the portion of the abdomen below the border of the liver. While in the great majority of instances the gall-bladder and its contents may be found located immediately under the border of the liver, in some instances the gall-bladder may be found as low as the ceco-ileac joint or the crest of the ilium, or anywhere in the right side in the right half of the abdomen, and if this area is not thoroughly investigated preceding the administration of the meal and while the bowels are thoroughly emptied, gall-stones will be overlooked in a number of instances.

The third and most important reason for not giving a meal preceding this study, is the fact that the duodenum, which is frequently adherent to the gall-bladder in gall-bladder disease, is so active in peristalsis that the peristaltic movement of the duodenum causes much movement of the gall-bladder and of any gall-stones which may be present, that the examination with reference to the gall-bladder would again be valueless.

Case, George, Cole and myself have determined that at least 50 per cent. of the gall-stones can be demonstrated and some valuable evidence obtained in about 25 per cent. of the cases; hence, this portion of the examination cannot justly be omitted. Following this gall-bladder study the patient is then given 2 to 3 oz. of barium sulphate in a pint of buttermilk, as originally described by me[11] in 1907. If the stomach and duodenum are found to be morphologically normal, the patient is then allowed to eat regular meals, in fact allowed to eat an immediate normal breakfast; and this opaque meal is then followed through the gastro-intestinal tract and studies made to determine any pathology that may be present. After the meal has been followed through the gastro-intestinal tract at least for forty-eight hours, the patient is then given an opaque enema and the general morphology and outline of the bowels determined.

THE PATHOLOGY ASSOCIATED WITH INTESTINAL STASIS DEMONSTRABLE BY THE ROENTGEN RAYS.

I. *Position of the Stomach and Bowel.*—I agree with Case that the position of the stomach and bowels, as only determined by the roentgen rays, is the least important of all the observations that we may make. There are no two noses alike, and there is no reason to expect two stomachs to be alike in shape or position. The shape and position of the viscera conform to the general shape of the body and to the habits of the individual. While making physiologic studies of the gastro-intestinal tract in 1907, I learned that a low position of the viscera was entirely consistent with health, my observation having been made upon a series of medical students who were healthy and attending to their usual college duties.

More recently Case[12] has examined 30 women students who believed themselves in normal health. In 27 out of the 30 the level of the transverse colon reached well below the navel, many of them down into the true pelvis. In only 3 was the transverse colon so high as to reach within an inch of the navel even with the patient lying supine. In only 3 out of the 30 did the lower border of the stomach reach so high a point as an inch below the navel with the patient standing. One of these 3 students was short and stout so that the stomach and transverse colon reached higher than usual. In the other 27 the lower border of the stomach averaged 2½ or 3 inches below the navel when the patient was standing. In general, the examination of stout patients shows the stomach and transverse colon to be high in the abdomen, while the examination of slender individuals shows the stomach and colon correspondingly low. We certainly have no right to choose an arbitrary condition for the lower border of the stomach or the lower border of the transverse colon. These vary with the individual and vary under different conditions in the same patient examined at different times. The position of any one of the abdominal organs only becomes important when it has lost its relation with regard to the other abdominal organs. One should note, however, the position of the pylorus, the cecum and the appendix, kidney and lower border of the liver when demonstrable.

II. *Kinks and Bands.*—Jordan, who is associated with Lane, has been the first among the roentgenologists and most persistent in calling attention to kinks which are produced by the various evolutionary bands: (*a*) The first of these bands occurs at the end of the first portion of the duodenum, resulting in a kinking, and some dilatation of the first portion of the duodenum and stagnation in the stomach; (*b*) at the duodeno-jejunal junction; (*c*) in the terminal portion of the ileum; (*d*) at the hepatic flexure; (*e*) at the splenic flexure.

I believe that entirely too much importance has been attached to these bands. That these kinks do occur, I believe cannot be denied, but that they are commonly the cause of symptoms, I believe remains to be proved. These kinks are demonstrable when present by examining the patient in the vertical and in the recumbent posture, and by palpating the opaque intestine under the fluoroscopic screen in such a way as to determine the points of attachment or traction; also by observing any delay in the passage of the contents of the bowel on the proximal side of the kink, which should also be associated with dilatation.

III. *Dilatation and Elongation.*—Elongation takes place first and dilatation only occurs after atony develops in the bowel. Elongation of the bowel may be present for a long time and be distinct evidence of pathology before dilatation develops, and if one only looks for dilatation of the gut one will often be deceived.

In the order of observation during the study of the gastro-intestinal tract, the first point of dilatation and elongation is in the duodenum, and especially the second portion of the duodenum, which has been especially emphasized by Jordan. Jordan says: "The normal length of this portion of the duodenum is from 2 to 3¼ inches, which in association with intestinal stasis the static duodenum often measures 4 to 5 inches, and more in severe cases." I have rarely seen any evidence of dilatation of the duodenum in connection with intestinal stasis. With regard to intestinal stasis causing duodenal ulcers, it cannot be confirmed by my observation, and this agrees with Bassler.[13] Generally speaking, the food passes more rapidly through the small bowel and large bowel in duodenal ulcer, and the stomach empties itself more rapidly than normal in connection with duodenal ulcer.

Elongation of the ileum cannot be determined because we have no fixed point from which to make our measurements. I have never observed distinct dilatation of the ileum excepting in patients upon whom there had been previously performed a colectomy after which a portion of the small bowel is forced to take up the functions of the colon which previously was present.

The cecum and ascending colon are frequently involved. Elongation is difficult to determine because of the curvatures in the lower end dipping deep into the pelvis, and the upper end curving backwards toward the hepatic flexure. Quite commonly in intestinal stasis the cecum and ascending colon are markedly dilated and frequently the cecum is found to lie deep in the pelvis.

Insufficiency of the ileocecal valve depeds upon the obstruction of the colon lower down. This obstruction leads to overdistention of the first portion of the colon—atony results; following this atony there is stretching of the ileocecal valve resulting in incompetency. Incompetency of the ileocecal valve may also be due to adhesions of an inflammatory type secondary to appendiceal disease. This same view is held by Case,[14] Kellogg,[15] Groedel,[16] Dietlen,[17] and others.

The transverse colon becomes very much elongated; I believe seldom dilated. Reduplications in this portion of the colon are frequently present. The descending colon frequently shows elongations and at times distinct loops are formed. These loops, while I believe they are secondary to the intestinal stasis, at times, as I have previously described, will give rise to very definite obstructions and symptoms.

The pelvic colon shows the most frequent elongation or redundancy. I called special attention to this condition in a paper read before the section on practice of medicine of the American Medical Association at its annual meeting in 1912,[18] describing it at that time as a "sigmoid loop." At that time I was uncertain as to whether this was a congenital elongation or the results of long distention. I feel convinced now that this "sigmoid loop," or elongation of the pelvic colon, is the result of repeated and long-continued distention; in other words, a draining backwards from too long retention in the rectum. I believe this elongation of the pelvic colon results from neglect of the rectal reflex. This may occur early in childhood, during adolescence, or during adult life, and may be the result of illness, modesty, procrastination, haste or business anxieties, as a result of which the individual is unwilling to give sufficient time for the evacuation of the bowels or may be the result of laziness or local disease. After a time this reflex is only excited by unusual accumulation or by unusually strong peristaltic waves moving downward from above.

As a result of this accumulation in the rectum, the fecal matter is dammed backward into the pelvic colon and the accumulation gradually stretches the pelvic colon until it may reach the diaphragm or the under surface of the liver. The accumulation of fecal matter, but more commonly of gas, as I have shown in a previous paper, may lead by direct pressure to obstruction in the transverse colon, to obstruction to the passage of food through the stomach, or may lead to the formation of a kink, and secondarily to obstruction, especially at the lower end of the iliac colon.

In the very excellent paper on the redundant sigmoid by Reed,[19] he claims that a sigmoid which is longer than 10 inches is pathological. At present the portion of the colon lying over the ilium is spoken of as the iliac colon. The portion of the colon between the lower border of the ilium and the upper portion of the rectum is now spoken of as the pelvic colon. Reed gives the length of the sigmoid as 10 inches. It is often very difficult to measure the length of the pelvic colon because it is in these conditions frequent in intestinal stasis, frequently coiled upon itself two or more times, and it would be utterly impossible to stretch it out sufficiently to measure it. This is especially true when adhesions are present which are quite common.

In describing the symptomatology of the redundant sigmoid, Reed says: "Probably the most constant symptom is constipation with more or less colicky pain. These colicky pains are more liable to precede than to attend efforts at defecation, although they may be experienced at both times. The constipation is liable to alternate with diarrhea, especially in cases of so-called mucous colitis with a large proportion of mucous contents in the stools."

There are generally more or less pressure symptoms in these cases, pain referable to pressure on the sacral plexus, generally more marked on the left side, but often present on both sides. Cystic irritations, generally without mucus in the urine, is rather a frequent complaint. In men there is often discomfort, distinctly referable to the prostate and obviously resulting from pressure on that gland. In women there is generally marked tenderness in the ovarian regions, especially on the left side. This may be due to pressure not only on the ovary itself, but also on the vessels and nerves within the broad ligaments. Other symptoms are pain at the hepatic and splenic flexure just under the margin of the ribs, in the left upper quadrant. Pain in the left upper quadrant is especially significant of angulation and consequent retardation at the splenic flexure.

It is my opinion that this elongation of the sigmoid or the pelvic colon is only a part of the general intestinal stasis, and all the symptoms referable to intestinal stasis may be found associated with this condition, and might be easily ascribed to and described in connection with elongation of the pelvic colon. I believe, therefore, that we should not regard this as an entity, but only as part of this general process. It results from a damming backward of the feces. The constant pressure from this accumulation below and the oncoming waves and contents of the colon above leads to this condition. I have found this elongation of the pelvic colon especially present in the epilepsy of childhood, and I believe that it is very significant. I know of some of these same little patients treated on the basis of intestinal stasis that have been at least improved with regard to their epilepsy if not totally relieved. Dilatation of the rectum is almost universally present in connection with intestinal stasis.

The demonstration of the elongation of the pelvic colon or sigmoid is best accomplished by a colonic injection or opaque enemata. This is given in the supine posture, and normally the pelvic colon is lifted out of the pelvis. When this cannot be done adhesions are commonly present. Further evidence of adhesions can be determined by manipulation of the colon under the fluorescent screen.

Case says: "The mesocolon is also very short at the pelvirectal junction, but the pelvic colon itself should be freely movable. In heavy patients it is not always possible to test the mobility of the pelvic colon by actual manipulation of its loop, but even in these cases accurate deductions can be drawn from a comparison of the height reached by the pelvic loop during the filling of the colon by an enema."

IV. *Adhesions.*—The adhesions which are of the most importance in connection with intestinal stasis are probably those associated with the pelvic colon, and are commonly produced by pelvic inflammation either connected with the ovaries or with the appendix. These adhesions may involve the pelvic colon or the terminal portion of the ileum and the cecum. They can be demonstrated or determined by manipulation of the various coils of the pelvic colon under the fluoroescent screen. (I must again call attention to the extreme danger to the operator in this manipulation of the colon under the fluoroescent screen. Much of this manipulation can, however, be done by means of a wooden spoon or distinctor, and the hand controlling this spoon can be held above the lead glass of the screen and be shielded by means of an opaque rubber glove.)

Gray,[20] in describing the effects of the mesosigmoid adhesion on the ovarian and spermatic veins, says: "These veins run directly under this adhesion and are often more or less caught up in it. If the sigmoid is loaded, and the patient is erect, there is too a direct drag or pressure on these veins, causing congestion of them below the point of application of the force. This in my opinion explains the aggravation of so-called ovarian neuralgia or the pain of a varicocele when the patient is constipated and the amelioration of symptoms when the patient lies down. Owing to the anatomical arrangement of the female pelvis, as compared with a male, deleterious effects are more marked in the former."

Adhesions may also be present about the cecum and ascending colon and gall-bladder region,[21] and may not only fix this portion of the gut to the abdominal walls, but may bind various coils to one another. Unless these adhesions distinctly cause evidence of obstruction or local painful irritation, I believe that they should not receive surgical attention. Adhesions may also bind the ascending and first portion of the transverse colon together, making what has been described as the double barreled gun. Three of the most marked cases of mechanical obstruction that I have seen have been due to extensive adhesions about the splenic flexure.

V. *Movements of the Bowel and Its Contents.*—As to the rate of movement of the contents of the bowel, there is considerable variation within normal limits, and the rate is influenced by the condition within the stomach, and the psychical and physical condition of the patient. Generally, however, the head of the opaque meal reaches the hepatic flexure by the end of six hours, and reaches the splenic flexure at the end of the ninth hour, and in twenty-four to thirty hours should have passed out of the rectum. In the great majority of instances the terminal coils of the ileum are still filled at the end of eight hours, but I believe this has no pathological significance. It may be very much prolonged by delay in emptying the stomach, due to some pathological condition about the pylorus. Only when the terminal coils of the ileum are filled at the end of twenty-four hours do I attach to it any special importance, and then I find that it is due to an incompetent ileocecal valve in the great majority of instances rather than to obstruction at the ileocecal valve.

The four types of movement recognized in the large bowel are peristalsis, anti-peristalsis, churning or segmentation, and the sporadic mass movement (first described by Holzknecht in 1909) and which is the chief factor in the forward propulsion of the colonic contents. The contents of the colon can be shifted very little by palpating even with strong pressure of the palpating hand. Groedel was unable to move the contents of the colon with a vibrator in full action. Anti-peristalsis is most active in the proximal portion of the colon up to the right and middle third of the transverse. At this point Cannon has described a constriction ring. I have observed a pronounced spasm of this constriction ring associated with gall-bladder disease.

VI. *Spastic Conditions.*—Retention in the ascending colon may continue for days even though the remainder of the bowel is emptied. I can agree with Case that this portion of the colon is kept filled by the reverse peristalsis, *i. e.,* the contents of the proximal portion of the colon is carried forward and meeting the spastic conditions is returned to the proximal portion of the colon again. I have, however, also observed this same retention in the first portion of the colon even when the patient has a regular bowel movement every day or when the patient is suffering from diarrhea. I am repeatedly called upon to examine patients on account of diarrhea when I find the real condition is an intestinal stasis involving the first portion of the colon. Retention in the first portion of the colon is also commonly present in association with the mucous colitis, and in these cases, by the roentgen rays, one can recognize the mucus lying in strings in the descending colon and pelvic colon, impregnated with barium.

Both the distention of the right half of the bowel and the degree of delay are exaggerated by the administration of common laxatives. Case says: "The cause of constipation in the majority of instances is located below the crest of the left ilium and, if not primarily due to spasticity, at least exhibits spasticity as an important factor." We formerly considered many cases of constipation as due to atrophy of the bowel muscles or atony. We now have learned that in the majority of cases the bowel is hypertonic. Later the distention in the right half of the bowel may give rise to atony of the cecum and ascending colon, so that we deal with a combination of the atonic and hypertonic conditions in so-called dyskinetic constipation, as Schwarz terms it. Rectal stasis or dysesthesia should be relieved by small enemata and not by laxatives. The high enema is a misnomer. Enemata should be used preceding operations rather than purgatives.

Case says: "The spasticity of the left half of the bowel may be recognized roentgenologically (*a*) by the appearance of isolated boluses in the left half of the bowel when the study is made following a barium meal. The more spastic the bowel the fewer and smaller the boluses. (*b*) A small quantity of barium enema will suffice to fill the colon as far as the splenic flexure. The right half of the bowel is usually dilated and the ileocecal valve incompetent, permitting the enema to flow backward into the terminal ileum.

VII. *Constriction and Neoplasm.*—Organic constrictions by means of bands in my experience are rare. Obstructions by neoplasms may be present for a long time and give rise to no other symptoms than those of constipation and intestinal stasis, and those of serious organic disease such as carcinoma of the rectum, sigmoid or some other portion of the colon. They might be allowed to develop over a long period of time before they are recognized unless the roentgen rays are employed early in the investigation. Especially should one be suspicious when a patient complains of bleeding piles associated with constipation. While in the majority of instances the patient's diagnosis is correct, I have seen repeatedly carcinoma as the cause of the symptoms, though neglected and undiagnosed over an abnormally long period of time.

CONCLUSION.

1. A careful roentgen study furnishes the most valuable evidence as to the actual pathological conditions present in the gastro-intestinal tract associated with intestinal stasis.
2. The most constant factors are rectal retention and secondarily retention in the pelvic colon with elongation of the pelvic colon and transverse colon, dilatation of the ascending colon, atony, and incompetency of the ileocecal valves, resulting in regurgitation into the ileum, and absorption of the contents.
3. A spastic condition of the bowels is present in the majority of instances, and this is aggravated by irritative laxatives, to which class most laxatives belong. The spasticity may be caused by appendiceal or gall-bladder disease.

BIBLIOGRAPHY.

[1] Lane (*Proc. Roy. Med. and Chir. Soc.*, London, 1913, Vol. VI, p. 94).

[2] Saundby (*Proc. Roy. Med. and Chir. Soc.*, London, 1913, Vol. VI, Part 1, p. 43).

[3] Hertz (*Proc. Roy. Med. and Chir. Soc.*, London, 1913, Vol. VI, Part 1, p. 164).

[4] Lane (*Brit. Med. Jour.*, November 1, 1913, Vol. II, p. 1,125).

[5] Johanbroeke (*Therap. Gozuti,* December 15, 1915, p. 848).

[6] Bainbridge (*Med. Rec.*, November 11, 1916, p. 672).

[7] Metchnikoff: Prolongation of Life, p. 69.

[8] Satterlee (*Amer. Jour. Med. Sci.*, 1916, pp. 727 to 738).

[9] Reed (*Med. Herald* (Kansas City), February, 1917).

[10] Chapple (*Brit. Med. Jour.*, April 22, 1911).

[11] Pfahler (*Jour. Amer. Med. Assoc.*, December 21, 1907, Vol. XLIX, pp. 2,069 to 2,074).

[12] Case (*Jour. Iowa State Med. Soc.*, February, 1916).

[13] Bassler (*Jour. Amer. Med. Assoc.*, October 24, 1914).

[14] Case (*Amer. Jour. Roentgenology*, July, 1914. *Internat. Abs. Surg.*, December, 1914, pp. 581 to 588).

[15] Kellogg (*Med. Rec.*, June 21, 1913).

[16] Groedel (*Fortschr. auf dem Geb. der Roentgenstr.*, 1913, Vol. XX, p. 192).

[17] Detlen (*Fortschr. auf dem Geb. der Roentgenstr.*, 1913, p. 21).

[18] Pfahler: Adhesions and Constrictions of the Bowel. (*Jour. Amer. Med. Assoc.*, November 16, 1912, Vol. LIX, pp. 1,770 to 1,774).

[19] Reed (*Jour. Amer. Med. Assoc.*, August 8, 1914, p. 449).

[20] Gray (*Brit. Med. Jour.*, January 24, 1914).

[21] Pfahler (*Jour. Amer. Med. Assoc.*, June 17, 1911, Vol. LVI, pp. 1,777-1,779).

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DISCUSSIONS.

John P. Sawyer, Cleveland, Ohio: I have been very much interested in the discussion, or symposium. I want to speak of how I was impressed by a remark of Dr. Smithies as to the fact that people will report to us, even if we take their histories repeatedly, that they have a daily normal movement, and this without knowing what a normal movement should be; that is, in the way that a history is usually taken. For a number of years, however, I have been putting to patients these questions, "What is a normal movement? What should a healthy individual discharge from the bowel?" I have asked this question of many hundreds of patients, and can recall only two or three who knew what a normal healthy movement should be. I think it would not be a bad thing, if we not only considered here the fact that we may be concerned with the late results of these long-existing constipations from childhood, but also the fact that the parents and even the grandparents, cannot tell what the children should have for a movement. We usually think that these results may be due to long-continued neglect, but they may be due also to ignorance; and we should extend a little knowledge to people as to what a normal bowel movement should be. People should be trained to have these movements.

My experience is in accord with Dr. Pfahler's remark on his very excellent pictures, that the bowel does become distended. I cannot say truly as to its elongation; but I have seen cases which presented similar appearances, but which, after proper treatment, no longer presented these appearances when examined. I feel that it is possible for this tissue, which is a physiologically equipped tissue, to become disturbed, and regain, through its power of recuperation, something of its normal condition. I think that we should emphasize very largely, and more widely than we do, attention to what is really a physiologically healthy movement of the bowel. Unless we take this into our practice and apply it in treatment, we shall be exemplifying the remark of Epictetus, that "the human understanding is such that if given two horns of a dilemma, it will usually take the wrong horn."

Dr. Jacob Kaufmann, New York City: I wish to endorse what Dr. Einhorn said on this topic, when he tried to define what is really meant by intestinal stasis, and for answer substituted the old definition of constipation. I think it is a perfectly correct conception, except in the cases which Dr. Lynch mentioned, in which we are dealing with the results of mechanical obstruction, which are an entirely different class of cases. Aside from these, the conditions usually described as intestinal stasis are nothing more than intestinal constipation.

Dr. Sawyer asks his patients, "What is a normal movement?" I think that there is no amount that may be considered normal. The amount passed by different individuals differs greatly. The amount of feces does not depend so much upon the amount of food taken as on the amount of the gastro-intestinal secretions, which varies greatly, and the amount of bacteria in the intestines. In some stools as much as one-third of the total amount of feces may consist of bacteria. Some people have a good solid bowel movement three times a day, and others get along with having one movement in three days or longer. When I was a young physician, one of the first patients I saw in private practice answered, when I asked, "Do your bowels move regularly?" "Yes, Doctor, quite regularly; every Friday." I examined her, and her bowels were comparatively empty. I had occasion to observe a colleague in New York, who was under the impression that, because he had rare bowel movements, he was suffering from intestinal obstruction, and was selecting food which like meat leaves small remnants. He used to have periods of two, three, and once even four weeks without a movement; and when I examined him after he had been without a movement for over 2 weeks I found his abdomen empty. By putting him on a diet, I got his bowels to act by having him take large quantities of vegetable food. He has had regular bowel movements ever since. I have known other people to go three or four weeks without a bowel movement. The question, what is stasis? does not depend on what is passed, so much as on what remains behind. Are we able to decide on that by X-ray examination? I doubt it. I think that the physical examination of the abdomen will show whether there is stagnation or not; and the mere form, width and length of the colon demonstrated by X-ray do not indicate whether there is stagnation or not.

I also agree with Dr. Einhorn that constipation does not necessarily produce these nervous symptoms, as he designated them, which are usually explained as auto-intoxication. I think that it is nearer the truth to take these as a concomitant element, and say that the constipation is something that goes with these nervous symptoms. In cases of migraine that have been thoroughly studied, in which the migraine comes on once in four weeks (in a woman, for instace, before menstruation), and in which an otherwise normal stomach suddenly becomes atonic, the atony is not because of the headache and vomiting, but as result of the general upset brought on by the imminent menstruation.

Dr. H. W. Soper, St. Louis: Dr. Einhorn made a distinction between a normal and diseased colon. I think that closer study of focal infections any place in the body will reveal the fact that the symptoms of focal infection are quite similar to those that we usually ascribe to intestinal stasis. Therefore, when confronted with one of those cases that appear to be due to intestinal stasis, we should first solve the problem as to whether we are dealing with a focal infection; and so, by a process of elimination, attempt to ascertain whether there is any infection outside the colon, and then direct our attention to infections in the colon itself. By so doing, I think that we shall find that our cases of intestinal stasis will gradually diminish in number, just as the cases termed "neurasthenia" are becoming less frequent in our case records.

Dr. Geo. W. McCaskey, Fort Wayne, Ind.: I feel some hesitation in attempting to discuss this subject, and do not feel quite sure of my own bearings regarding many of the questions involved. I think that I occupy a middle position between some of the extreme positions that have been brought out, by the surgeon, and by the internist. Just because an individual, here, there or elsewhere, can go for a week, two weeks, or three weeks without having a movement, and without symptoms, does not seem to me to prove that it is a normal physical condition. I think we know that it is not. Nor does it prove, on the other hand, that that very delay, or stasis, if you please to call it such, that constipation, if you prefer the name, may not, and does not in many cases, produce very severe symptoms. I am fully convinced, on the ground of clinical observation, that we often have to deal with an intestinal auto-intoxication, which may be the result of this stasis. On the other hand, I think that it is equally clear that we have that intestinal auto-intoxication in many cases, even more severely when the bowels are moving too freely and the intestinal contents are too fluid. Bacterial growth is rife. Bacterial toxins are produced abundantly, and absorbed to a certain extent by the intestinal tract. So the question is an exceedingly complex one, and anything approaching generalization is dangerous, or difficult, at least.

Taking, now, the cases of stasis, so-called, it should not be difficult to tell how long it takes for the food to pass through the bowel. There is no regular schedule for this. Individuals may be in perfect health and have very different schedules, but it is a simple matter to find out what the schedule is. Give a charcoal tablet to-day, and a carmine capsule to-morrow, and you can easily tell how long it takes the material to pass. Suppose it be shown that it takes four or five days. If the patient is well, this may be his usual schedule. Then let him alone. But if he has those nervous manifestations, the case should be studied from the other angle: and it must be considered probable that the symptoms are the result of intestinal derangement. In a certain proportion of cases, I believe that this condition is responsible for the symptoms attributed to it; but in many cases, this is not so at all. When we have the concomitant symptoms that we think may be the result, the important question is, What are we going to do? A certain portion of these cases are surgical. Here the X-ray helps to differentiate the cases that should be treated surgically (and there are probably many of these) from those that the surgeon should not touch, and that surgeons like Dr. Gibbon will not submit to any radical procedure. So we simply treat them along the line spoken of by Dr. Einhorn. We must try to discover, so far as we can, the bacterial process that may cause various infections. Whatever will lower the tonicity and physical integrity of the intestines will aggravate the condition. These cases may be exceptional, but I am inclined to think that they are more the rule. In certain cases, we shall find that as we proceed along these lines and the bowel movements, through exericse, fresh air, better nutrition, proper diet, etc., that the symptoms subside, and the patient is benefited, the bowels moving regularly. I have seen this happen again and again. The symptoms, in these cases, were due to some sort of intestinal intoxication, the result of stasis or constipation, or whatever you please to term it.

DR. PFAHLER, closing: Regarding the question about diverticulitis. I will answer that in connection with the paper on diverticulitis.

I should like to call attention to a view expressed by Dr. Hertz in a discussion before the Royal Society, in 1913, that sometimes patients have more symptoms after taking a purge than while they are definitely constipated and are having no bowel movements. That fact was explained by him on the ground that in giving a purge we render the contents of the bowel more liquid and make toxin substances more easily absorbed. I believe that it is not a question of how many bowel movements, but of the symptoms produced by the absorption of these toxins.

THE MEDICAL TREATMENT OF CHRONIC INTESTINAL STASIS.*

By Max Einhorn, M. D.,
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Intestinal stasis, according to Lane, means a delay in the passage of the intestinal contents, resulting in the absorption into the circulation of toxic or poisonous material. This condition occurs principally in visceroptosis, with kink formation.

Before discussing my topic of medical treatment, it is essential to have a clear understanding of what is really meant by intestinal stasis, leaving theories aside. Delay in the passage of the intestinal contents is of frequent occurrence, and is usually designated as "constipation." Absorption of toxic material from the intestine into the circulation is a presupposed event—and its existence is taken for granted by Lane and his followers, if there appear disturbances of the nervous system accompanying the constipation. As such, there may be dizziness, lack of concentration of the mind, headaches, sleeplessness, anorexia, flatulence, weakness, etc. There is no doubt that these symptoms are frequently encountered in association with constipation, but the claim that they are toxic phenomena due to absorption of poisonous material from the intestine is a mere theory, which has not been proven.

In order to obviate any prejudice, I shall substitute for intestinal stasis "constipation with nervous phenomena," which signifies the same condition without giving a definite theory.

Regarding the etiology of nervous disturbances, the following explanation appears quite plausible. Health requires a proper balancing of work and rest, evenly divided among the various organs of the body. Overstrain leads to disturbances either of a general character involving the entire organism or of some parts in the vicinity of the affected spot. These disturbances are usually caused by the regulatory mechanism of the body and serve as defense measures. Too much work creates fatigue, which compels the body to rest. During the periods of relaxation all the waste products manufactured by the strained work are eliminated from the system. After this the organism is in proper balance again and euphoria exists. If, however, the overstrain of one organ, or group of organs, goes on uninterruptedly for quite a long time without sufficient relaxation to reestablish the proper equilibrium, there is a gradual accumulation of waste products in the organs concerned, or there exists an insufficient elimination of the waste material which gradually increases in quantity. The organism, working under great strain and pressure, overcomes the difficulties for a while, but ultimately the wants of the body make themselves felt, and the patient collapses from utter fatigue. He is then sick and must rest. Thus a chance is obtained for a period of relaxation—and complete elimination. Worry, anxiety, too much mental work—especially in individuals with a slightly impaired constitution—are apt to produce migraine and other nervous symptoms.

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*Read before the American Gastroenterological Association, on April 30, 1917, at Atlantic City, N. J.

Elimination of waste products takes place by way of the lungs, kidneys, skin, liver, and the digestive apparatus. In constipation, elimination through the digestive tract is partly retarded, thus favoring neuropathies. The latter again, by inhibiting intestinal peristalsis, enhances constipation. In this way there is a constant circulus vitiosus, the complete removal of which is not an easy task.

Some will ask what difference there is between assuming that the body is overburdened with waste products and deficient elimination or that the body is troubled with autointoxication from the intestine. My answer would be that there is a vital difference between these two hypotheses, especially involving the mode of treatment. Intestinal autointoxication ascribes the main source of the evil to the colon. As a result, this organ bears the brunt of the blame. The surcharge of waste theory ascribes the fault to many organs—especially the central nervous system—and the therapy will have to be directed toward improving the general state of the organism, in conjunction with the bowel difficulty.

Let me now outline the treatment of chronic intestinal stasis or constipation with manifold neurotic symptoms. Instead of frightening the patient with autointoxication theories, he must be assured that *no* harm will result from the lack of a bowel movement for a few days. He must be instructed to go to the toilet every morning at about the same hour, and not to think or worry about the bowels during the rest of the day. Nerve sedatives are occasionally helpful. Ample food and a great variety of it should be given. Vegetables, fruits, and salads should play a prominent part in the dietary of the patients. Water should be taken liberally at meals and also between meals. If these measures alone are not sufficient mild aperients may be employed, as, for instance, cascara segrada, rheubarb, phenolphthalein, etc. Agar-agar helps to lubricate the fecal matter and is in itself useful in facilitating the bowel peristalsis. Phenolphthalein-agar as well as rheubarb-agar act especially well and can be used profitably for long periods of time. Olive oil and liquid petrolatum, in tablespoonful doses, taken when arising and retiring are likewise beneficial.

Cases in which abnormal conditions with regard to gastric secretions or to position of stomach and bowel exist have to be managed according to the generally known principles. This alone will frequently be of help in overcoming the constipation.

Saline enemas, likewise injections into the bowel of a small quantity of olive oil or water at bedtime—to be retained over night—are helpful in restoring the bowel function.

Abdominal exercises, massage of the abdomen, electricity applied percutaneously over the abdomen, or one electrode over the abdomen and the other in the rectum, are likewise beneficial measures which can be resorted to in obstinate cases.

Operations are seldom, if ever, required.

Of course I do not include in this group cases of constipation with real organic lesions interfering with intestinal prochoresis —stenosis or intussusception of the intestine, etc.—which give an entirely different disease picture and in which surgery plays the most prominent part.

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HIGH GASTRIC ACIDITY.*

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As definite statements as to maximum and minimum titrations of chyme are comparatively small in number, the following cases are recorded, to aid in establishing the standards of what may be expected. Except as stated, the figures apply to test meals consisting of about 50 grams of bread, 5 of butter and 200-250 c.c. of water, extracted 1-1¼ hour P. C., titrated by decinomal alkali with phenolphthalein for total acidity and with dimethylamidoazobenzol for free HCl, taking the latter reading at the change of color to orange, not at the final straw yellow. No account is taken of cases yielding less than 100 ° of total acidity, unless the free HCl acidity was over 50°, nor of cases of high total acidity due to fermentation in the absence or marked reduction of HCl.

	Total	Free HCl.	
G. A. McG......	99°	64°	6 hours after malted milk, 875 c.c. extracted, symptoms suggested hypochlorhydria with fermentation.
G. A. D........	122°	95°	
F. J. O........	104°	30°	
F. J. O........	100°	45°	
A. McW........	87°	58°	
J. C. R.........	116°	64°	
G. A. W........	102°	78°	
E. S...........	94°	70°	
J. J. McS.......	105°	40°	3 hours after beef, bread and butter and milk.
A. M..........	104°	15°	
G. H. G........	100°	64°	
H. L...........	146°	47°	
Mrs. Jas. A.....	100°	36°	
E. S...........	120°	47°	

It is scarcely necessary to call attention to the fact that a high total acidity is usually largely due to fermentation and, hence, occurs in chyme low in HCl that has not been promptly examined but has stood at room temperature, especially in summer. While hyperchlorhydric stomach contents do not usually develop any marked increase in acidity, even by long standing at room temperature, occasional instances are noted and, stagnation for long periods in the stomach, may also develop considerable fermentation acidity, in spite of the presence of considerable HCl, as in the case of G. A. D. This case is, to the best of my recollection, the record either for relative amount of HCl (95°) of absolute quantity when we consider the large amount of chyme. 150° may be considered as practically the maximum titration figure that will be encountered in clinical chemistry of any kind. I have several times found urine considerably over 100° but never as high as 150° and do not recall chyme over 150°, even with every opportunity for fermentation, with the exception noted subsequently. Curiously enough, in achylia, there is very rarely a tendency to fermentation within the stomach, the total acidity usually being not over 10.° Such chyme does not even seem to be especially liable to develop high fermentation acidity on standing. The lower limit of hyperchlorhydria may be taken at the approximate physiologic standard of 25°, corresponding to a 50 per cent. dilution of gastric juice of 50° HCl. The definition of hyperchlorhydria as beginning at the normal standard is justifiable when the absolute amount of chyme is high, especially when it is greater than the volume of the test meal; perhaps even when the symptomatology corresponds to that of cases with a genuine increase of HCl. Unfortunately, a good deal of the literature on hyperchlorhydria is based on qualitative tests, titration of total acidity, on the inclusion of cases that are barely euchlorhydric but rather cases of slightly reduced HCl secretion with factors favoring fermentation. Again, the measurement of HCl in degrees is often exaggerated 5-10° by reading at the final discharge of color instead of at the change from cherry red to orange.

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A. McW., August, 16, 1904, had a total acidity of 84°, 37° of free HCl. On standing 3 days, the results were 84° and 49°. May 29, 1905, the total acidity was 87°, free HCl 58°. Nineteen days later, the total was 95°, free HCl 57°; 36 days from May 29, the results were 96° and 52°, using the same chyme that had been kept at room temperature without any attempt at preservation.

A. H. S., January 6, 1905, had a total acidity of 84°, 37° of free HCl. Seven days later, the same chyme was read at 113° total and 44°

free HCl. These changes, due to standing, are about what may be expected. When increases in free HCl readings occur, they are probably due to displacement of combined HCl, or, sometimes perhaps to action of rather strong organic acids like mineral acids.

A very marked and entirely unprecedented change of acidity occurred in the case of J. J. McS., previously listed. April 13, one hour after two slices of bread and butter and a glassful of water, 175 c.c. of chyme was obtained, the centrifuged filtrate amounting to 60 per cent. of the total volume. Total acidity 71°, free HCl 26° (total discharge of color with dimethyl 37°, alizarin end-point 50°). No starch but erythrodextrin and sugar both present. The unfiltered chyme yielded practically no gas after 20 hours' incubation at 37°. The filtrate from the incubated chyme gave a total acidity of 99°, the free HCl was read at 35°, the final discharge of color with dimethyl being at 49°. Meantime, the filtrate left standing at room temperature increased in total acidity to 78°, the free HCl being unchanged, but the final discharge of color with dimethyl being raised to 49°. These results are all within the usual limits and are stated merely for comparison with the examination of April 29, listed in the first table.

This latter examination was made on 125 c.c. of chyme obtained 3 hours after a meal of beef, bread and butter and milk, with special view of securing information as to the duration of the acid excess. Total acidity 105°, free HCl 40°, final discharge of color with dimethyl 53°, changed after 20 hours' incubation to 114°, 30° and 47° respectively. Meantime the filtrate which had stood at room temperature, gave the astonishing results of 226° total acidity, 158° HCl and 172° final discharge of color with dimethyl. It was supposed that, inadvertently, 20 c.c. instead of 10 c.c. of filtrate had been deposited in the titration dish but a check test gave 189° for the alizarin end-point and 229° total acidity, 158° and 175° for the dimethyl titrations, well within the error of readings in general. It was then thought that, in some way, the alkaline solution had been weakened but this was shown not to be the case. The only other explanation of an adventitious error that occurred to the writer at the time or subsequently, was that acid had accidentally been spilled into the filtrate left standing, but this was impossible so far as could be determined by the circumstances. How to explain these astonishing changes in acidity, I do not know and they are reported with the hope of securing light on the question.

I have at various other times described a method of approximate determination of the gastric acidity by auscultíng while a concentrated solution of sodium bicarbonate is being swallowed.

The effervescence is easily detected. This test is mainly of value when it results negatively, but it has some quantitative value obtainable after considerable experience. Usually 5 grams (about a teaspoonful of sodium bicarbonate) more than suffices to neutralize the chyme unless it is quite highly acid. For example H. L., noted in the first table as giving a maximum total acidity of 146° and free HCl to the amount of 47°, other examinations being high but not extraordinary, on two or three occasions took 2 teaspoonfuls of soda before the effervescence subsided. A. McW., also listed in the first table, had a hyperchlorhydria lasting intermittently for at least 12 years. He was an out of town patient and opportunities for close examination were not very favorable. Last year, 12 years after the first demonstration of hyperchlorhydria, and 11 after the high result noted in the table, I saw him at midnight at his home in the country. It was not feasible to intubate, carry the chyme 50 miles for examination and return, as he was in great distress. Therefore, both for diagnostic and therapeutic purposes, he was given sodium bicarbonate which produced brisk effervescence. This was repeated after a few minutes, with the same result. As he had then taken about 10 grams of soda, it occurred to me to make a rough estimate of the amount necessary to neutralize the stomach contents. Just about 30 grams more were given, rather slowly, effervescence continuing steadily for 7 minutes. With the cessation of effervescence, relief was obtained and, except for favorable reports as to general condition and symptoms, I have not heard from the patient for over a year. He is taking magnesia occasionally as symptoms of superacidity occur. It is, of course, impossible to state from such an observation, the degree of total and hydrochloric acidity nor the volume of chyme, but it is obvious that the total amount of acids present, by volume or proportionate strength or both, must have been enormous—approximately 8 times that encountered in an average, moderate case of hyperchlorhydria.

DISCUSSIONS.

Dr. J. C. Hemmeter, Baltimore, Md.: The paper of Dr. Benedict is of interest because the clinician must have a feasible method, and Töpfer's method has forced its way into every clinician's laboratory. Therefore, it is essential for us to know how reliable it is. I know Töpfer personally. He has somewhat improved his method, but it will never give absolutely accurate results. He admits that it is correct only within certain limitations. In a normal stomach, the entire total acidity is due to hydrochloric acid. There is no reason why there should be anything else there. Otto Cohnheim regards the total acidity as due to hydrochloric acid. I cannot explain these discrepancies found by Dr. Benedict.

DR. ALLEN ARTHUR JONES, Buffalo, N. Y.: I should like to ask whether there was any reaction of nitric acid in the third and fourth titrations, after standing at room temperature.

DR. BENEDICT, closing: I would say, regarding the unreliability of the Töpfer method, that I have gone over several methods. They have a simplified silver and incineration method in Paris, but you cannot get away from titration finally. I have found, in general, that the results are fairly consistent; although there is a moderate margin of error. I regard these as high totals for a normal or excessive amount of hydrochloric acid. In this one, with 47° of free hydrochloric acid, we would expect a total acidity of 70 or 80°. That covers a large amount of variations. These cases can all be considered clinically as hyperchlorhydria without evidence of ulceration or fermentation, and no gas to amount to anything; and with one exception, no case of achylia. (?)

I should have to refer to my original notes, in order to answer the questions about nitric acid. (?)

DR. FRANK SMITHIES, Chicago, Ill.: To those interested in the inaccuracy of titration and test meal analyses, I would refer an article by Panton and Tidy published in the *British Quarterly Journal of Medicine* about three years ago, and containing a complete discussion of this whole matter.

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